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SULFANILAMIDE IN THE TREATMENT OF EXPERIMENTAL STREPTOCOCCIC AND PNEUMOCOCCIC MENINGITIS

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The remarkable success of sulfanilamide (para-aminobenzenesulfonamide) and its derivatives in the treatment of experimental and human infections due to beta hemolytic streptococci, and to some extent of those due to pneumococci, has given rise to the hope that these preparations will prove effective in the treatment of streptococcic and pneumococcic meningitis, especially in view of the high mortality of these diseases (as high as 97 per cent in the case of streptococcic meningitis, as shown in a recent survey by Gray [1]).

In February 1936 Caussé, Loiseau and Gisselbrecht [2] reported the recovery of a man with streptococcic meningitis of otitic origin following the intravenous and oral administration of a derivative of sulfanilamide (prontosil). The experimental work reported here was begun in January 1937, immediately after the publication of the paper by Long and Bliss [3] on the remarkable effects in the treatment of mice with septicemia due to beta hemolytic streptococci. Since then Smith and Coon [4] have reported the recovery from streptococcic meningitis of 2 patients, Schwentker and his colleagues [5] the recovery of 3 patients

From the Research Institute of Cutaneous Medicine.

1. Gray, H. J.: Streptococcic Meningitis: Report of a Case with Recovery, J. A. M. A. **105**:92 (July 13) 1935.

2. Caussé, Loiseau and Gisselbrecht: Méningite purulente otogène à streptocoques hémolytiques, traitée exclusivement par un azoïque. Guérison, Ann. d'otolaryng., February 1936, pp. 194-199.

3. Long, P. H., and Bliss, E. A.: Para-Amino-Benzene-Sulfonamide and Its Derivatives: Experimental and Clinical Observations on Their Use in the Treatment of Beta-Hemolytic Streptococcic Infection; Preliminary Report, J. A. M. A. **108**:32 (Jan. 2) 1937.

4. Smith, H. B., and Coon, E. H.: Meningitis Due to Hemolytic Streptococcus: Report of Two Cases with Recovery After Use of Prontosil and Sulfanilamide, Arch. Otolaryng. **26**:56 (July) 1937.

5. Schwentker, F. F., and others: Use of Para-Amino-Benzene-Sulphonamide or Its Derivatives in the Treatment of Beta Haemolytic Streptococcal Meningitis, Bull. Johns Hopkins Hosp. **60**:297 (April) 1937.

and Anderson [6] and Arnold [7] each the recovery of 1 patient following the administration of sulfanilamide or its derivatives.

So far there have been less hopeful results in the treatment of pneumococcic meningitis with these compounds. Schwentker [8] has reported on the treatment with sulfanilamide of 5 patients. One, with infection due to the type X pneumococcus, recovered. In a second, infected with type III, the spinal fluid became sterile after about five days of treatment, but the patient died three days later. The 3 remaining patients had some prolongation of life, since they died in seven, eight and nine days, but the spinal fluids did not become sterile.[9]

In view of these results and since there have not been any reports on the treatment of experimental streptococcic and pneumococcic meningitis with sulfanilamide and its derivatives, we thought it advisable to report briefly on this phase of the subject and to include what information we have been able to obtain concerning the mode of action of sulfanilamide on these experimental infections.

EXPERIMENTAL MENINGITIS

Rabbits and monkeys were employed and were infected by intracisternal inoculation.[10]

The culture of beta hemolytic streptococci used was that employed in this institute for several years in the production of experimental infections in rabbits by intradermal and intravenous inoculation.[11] The intracisternal injection of about 0.3 cc. per kilogram of weight of a 1:30 dilution of eighteen-hour hormone broth culture invariably produced meningitis with septicemia, which was fatal in about twenty-four hours, while the injection of 0.3 cc. per kilogram of a 1:60 dilution produced suppurative meningitis with septicemia fatal in from forty-eight to ninety-six hours.

Symptoms of meningitis were usually apparent about four hours after inoculation, at which time the spinal and the cisternal fluid were already cloudy with polymorphonuclear pus cells and streptococci. At the end of from twenty-four

6. Anderson, E. D.: Hemolytic Streptococcus Meningitis, J. A. M. A. **108**: 1591 (May 8) 1937.

7. Arnold, J. G.: Treatment of Hemolytic Streptococcic Meningitis with Para-Amino-Benzene-Sulfonamide, Ann. Int. Med. **10**:1198 (Feb.) 1937.

8. Schwentker, F. F.: Personal communication to the author.

9. Since the preparation of this article, Joseph Millet (The Intrathecal Use of Prontosil Soluble, J. A. M. A. **109**:2138 [Dec. 25] 1937) has reported that the intrathecal administration of 60 cc. of a derivative of sulfanilamide (prontosil soluble) had a marked bactericidal effect on type III pneumococcic meningitis in a patient who died of a type III pneumococcic bacteremia. Postmortem examination revealed no apparent chemical injury of the brain due to the drug.

10. All intracisternal punctures were conducted with the animal under ether anesthesia.

11. Kolmer, J. A., and Rule, A. M.: Experimental Streptococcus Infections in Rabbits for Therapeutic Investigations, J. Lab. & Clin. Med. **22**:1097 (Aug.) 1937.

to forty-eight hours the symptoms and signs of meningitis were more pronounced, with extreme prostration and highly purulent cerebrospinal fluid showing large numbers of streptococci in direct smears and cultures.

Sections of the spinal cord and the meninges showed the usual histologic changes of acute leptomeningitis, with hyperemia, edema and polymorphonuclear infiltration,

Fig. 1.—Section of the spinal cord and meninges of a rabbit which died ninety hours after the intracisternal inoculation of 0.3 cc. per kilogram of a 1 : 60 solution of eighteen-hour hormone broth culture of beta hemolytic streptococci (× 115).

which were particularly pronounced in the rabbits which survived for from seventy-two to ninety-six hours (fig. 1).

Cultures of the heart blood of untreated controls were invariably positive. This indicates that the experimental infection was one of fulminating meningitis with associated septicemia and was admittedly very severe for chemotherapeutic purposes.

Adult monkeys (Macacus rhesus) possessed a much higher natural resistance to this streptococcus, since the intracisternal injection of as much as 2 cc. per

kilogram of eighteen-hour hormone broth culture was required for the uniform production of suppurative meningitis fatal in about seventy-two hours. Symptoms and signs of meningitis were usually present about six hours after inoculation and pronounced in about twenty-four hours, with purulent cerebrospinal fluid showing large numbers of streptococci in direct smears and cultures. Septicemia, however, did not occur, as cultures of the heart blood were invariably sterile.

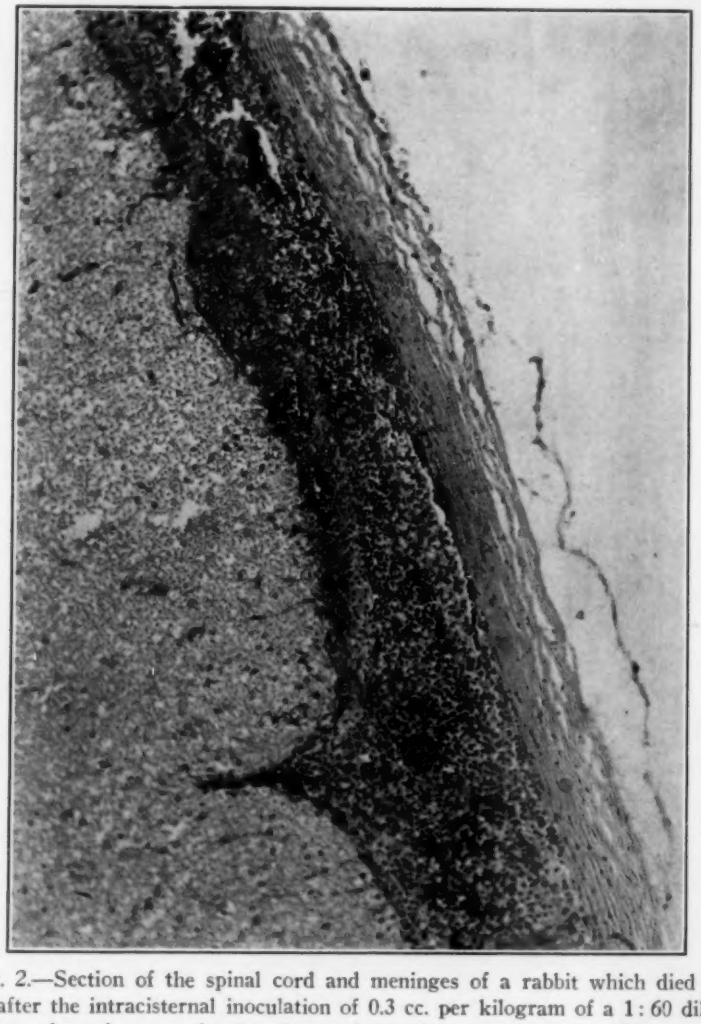

Fig. 2.—Section of the spinal cord and meninges of a rabbit which died sixty hours after the intracisternal inoculation of 0.3 cc. per kilogram of a 1:60 dilution of eighteen-hour hormone broth culture of type I pneumococci (× 122).

Histologic examination of the cord and meninges showed suppurative leptomeningitis, particularly marked in the upper end of the cord and similar to that observed in rabbits. Because of the longer duration of life and the absence of septicemia, these experimental lesions were much more favorable for treatment experiments.

Types I, II and III pneumococci were employed. The intracisternal inoculation of rabbits with 0.3 cc. per kilogram of a 1:60 dilution of eighteen hour hormone broth culture of type I invariably produced symptoms of meningitis in about four hours, with extreme prostration and cloudy cerebrospinal fluid containing large numbers of pus cells and numerous pneumococci in direct smears and cultures. Death followed in from twenty-four to forty-eight hours. Sections of the spinal cords and meninges showed acute leptomeningitis similar to the histologic changes produced by the hemolytic streptococcus; it was particularly well marked in the upper portion of the cords of those rabbits which survived for about seventy-two hours (fig. 2). As a general rule, however, the degree of inflammation was somewhat less severe than that produced by the strain of streptococcus.

Types II and III in doses of 0.3 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth cultures were somewhat less virulent, as the untreated controls survived for two, three or four days.

Cultures of the heart blood were likewise positive with all three types, so that the infection was meningitis with associated septicemia.

TABLE 1.—*Prontosil and Sulfanilamide in the Treatment of Streptococcic Meningitis in Rabbits* *

		Administration,† Gm. per Kilogram			
No.	Compound	Intramuscular	Intracarotid	Intracisternal	Results§
6	Prontosil solution	0.0025	.....	.....	All died in from 30 to 46 hr.
6	Prontosil solution	0.0125	.....	.....	All died in from 48 to 96 hr.
8	Prontosil solution	.....	0.025‡	.....	All died in from 30 to 48 hr.
6	Sulfanilamide	0.04	.....	0.016	All died in from 36 to 54 hr.
8	Controls	.....	.....	.....	All died in from 18 to 24 hr.

* Infected by the intracisternal injection of 0.5 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth culture of hemolytic streptococci.

† Four, eight and twenty-four hours after inoculation.

‡ A dose of 0.0125 Gm. in each common carotid artery, totaling 0.025 Gm. per kilogram.

§ All spinal fluids were highly purulent, with positive cultures; cultures of the heart blood were positive.

Types I, II and III were also used for the infection of M. rhesus monkeys, the intracisternal injection of 0.3 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth culture producing fulminating meningitis with septicemia (positive heart blood cultures) in from twenty-four to forty-eight hours.

RESULTS IN THE TREATMENT OF STREPTOCOCCIC MENINGITIS

In the first experiment, summarized in table 1, all of 8 untreated controls died from eighteen to twenty-four hours after intracisternal inoculation.

The intramuscular injection of a 2.5 per cent prontosil solution[12] in doses of 0.0025 Gm. (0.1 cc.) per kilogram, given four, eight and twenty-four hours after infection, slightly prolonged the lives of 6 animals. A dose of 0.0125 Gm. (0.5 cc.) per kilogram, administered at the same intervals, definitely prolonged the lives of 6 additional animals, since all survived from forty-eight to ninety-six hours after infection. Eight additional animals were given 0.0125 Gm. (0.5 cc.)

12. The name prontosil has been applied to several different preparations. The one we used was the disodium salt of 4-sulfamidophenyl-2'-azo-7'-acetylamino-1'-hydroxynaphthalene-3',6'-disulfonic acid.

in each common carotid artery,[13] at the same intervals, so that the dose totaled 0.025 Gm. (1 cc.) per kilogram, the lives of these animals were likewise slightly prolonged, as all survived from thirty to forty-eight hours after infection.

Sulfanilamide[14] in doses of 0.04 Gm. given intramuscularly and 0.016 Gm. given intracisternally per kilogram of weight, administered four, eight and twenty-four hours after infection, prolonged the lives of 6 rabbits, which survived from thirty-six to fifty-four hours after infection.

In the second experiment, summarized in table 2, the rabbits were inoculated intracisternally with a smaller amount of culture, which permitted the 4 untreated controls to survive about forty-eight hours.

Sulfanilamide was administered four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after infection, the doses and routes of administration being shown in the table.

Of 4 rabbits which received 0.1 Gm. per kilogram orally, 2 survived, and the remaining 2 died from forty-eight to fifty-four hours after infection.

Of 4 animals which received 0.04 Gm. per kilogram by subcutaneous injection, 2 survived, and the remaining 2 died about fifty hours after infection.

TABLE 2.—*Sulfanilamide in the Treatment of Streptococcic Meningitis in Rabbits* *

	Administration,† Gm. per Kilogram				Died			
No.	Oral	Subcutaneous	Intracisternal	Recovered	No.	Duration	Spinal Fluid‡	Blood‡
4	0.1	....	.....	2	2	48-54 hr.	+	+
4	...	0.04	.....	2	2	50 hr.	+	+
4	...	....	0.008	None	4	30-36 hr.	+	+
4	...	0.04	0.008	2	2	72 hr.	+	+
4	0.1	0.04	0.008	3	1	96 hr.	+	+
4	Controls.....			None	4	48 hr.	+	+

* Infected by the intracisternal inoculation of 0.3 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth culture of hemolytic streptococci.
† Four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation.
‡ Cultures positive.

Four animals given 0.008 Gm. per kilogram by intracisternal injection died from thirty to thirty-six hours after infection.

Of 4 animals given 0.04 Gm. subcutaneously and 0.008 Gm. intracisternally per kilogram, 2 survived and 2 died (seventy-two hours), the results being essentially the same as those observed in the 4 animals which received this dose subcutaneously.

Of 4 animals given 0.1 Gm. orally, 0.04 Gm. subcutaneously and 0.008 Gm. intracisternally per kilogram, 3 survived and 1 died (about ninety-six hours). This series therefore gave the best results, apparently because of the simultaneous administration of the compound by the oral and the subcutaneous route.

Better effects were observed in the treatment of streptococcic meningitis in monkeys, as shown in the results of an experiment summarized in table 3. Since these animals did not have associated septicemia, the experimentally produced meningitis was more satisfactory than that

13. Kolmer, J. A.; Rule, A. M., and Madden, B.: Chemotherapy and Serum Therapy of Pneumococcus and Streptococcus Meningitis, Arch. Otolaryng. **9**:509 (May) 1929.

14. Supplied by the Dermatological Research Laboratories, Philadelphia.

produced in rabbits for studying the effects of treatment with sulfanilamide.

Two untreated controls died about seventy-two hours after infection.

Sulfanilamide was administered twenty-four, forty-eight and fifty-four hours after infection to the treated animals, the doses and routes of administration being shown in the table. The infection was given a longer start before treatment was begun, since the meningitis was less fulminating than that observed in the rabbits.

Of 2 monkeys given 0.1 Gm. per kilogram orally, 1 survived indefinitely, and the life of the second was prolonged about eighty hours after infection.

Two which received 0.04 Gm. per kilogram by subcutaneous injection survived indefinitely.

Two which received 0.008 Gm. per kilogram by intraspinal injection died in from forty-eight to seventy-two hours.

Two given 0.1 Gm. orally and 0.04 Gm. subcutaneously per kilogram lived indefinitely. However, 2 which received 0.04 Gm. subcutaneously and 0.008 Gm.

TABLE 3.—*Sulfanilamide in the Treatment of Streptococcic Meningitis in Monkeys* *

	Administration,† Gm. per Kilogram				Died			
No.	Oral	Subcutaneous	Intraspinal	Recovered	No.	Duration	Spinal Fluid‡	Blood‡
2	0.1	....	.....	1	1	80 hr.	+	—
2	...	0.04	.....	2	None	0	0	0
2	...	....	0.008	None	2	48-72 hr.	+	—
2	0.1	0.04	.....	2	None	0	0	0
2	...	0.04	0.008	None	2	54-72 hr.	+	—
2	0.1	0.04	0.008	2	None	0	0	0
2	Controls.....			None	2	72 hr.	+	—

* Infected by the intracisternal injection of 2 cc. per kilogram of an eighteen hour hormone broth culture of hemolytic streptococci.
† Twenty-four, forty-eight and fifty-four hours after inoculation.
‡ A plus sign indicates a positive culture and a minus sign a sterile culture.

intraspinally per kilogram died at about the same time as the untreated controls (from fifty-four to seventy-two hours).

Two which received 0.1 Gm. orally, 0.04 Gm. subcutaneously and 0.008 Gm. intraspinally per kilogram lived indefinitely, the results apparently being due to the larger total dose administered

In view of the severe meningitis with associated septicemia observed in the untreated control rabbits and the severe meningitis produced in untreated control monkeys, resulting in death about seventy-two hours after intracisternal infection, the results of treatment with sulfanilamide are interpreted as very encouraging, especially when the compound was given orally or by subcutaneous injection or by a combination of the two methods. This was particularly true in the case of monkeys. The intracisternal or the intraspinal administration of sulfanilamide alone in a dose of 0.008 Gm. per kilogram (equivalent to 1 cc. of a 0.8 per cent solution per kilogram) was without apparent therapeutic effects in both rabbits and monkeys. The results observed with the oral administration of 0.1 Gm. per kilogram were probably not quite as favorable as those observed with the subcutaneous injection of 0.04 Gm. per kilogram.

RESULTS IN THE TREATMENT OF PNEUMOCOCCIC MENINGITIS

As shown in tables 4, 5 and 6, untreated control rabbits inoculated intracisternally with type I pneumococci died in from twenty-four to forty-eight hours of fulminating meningitis and associated septicemia; type II produced death in from forty-eight to seventy-two hours and type III in from two to four days.

Sulfanilamide was administered four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after infection, the doses and routes of administration being shown in the tables.

Four rabbits infected with type I and treated with sulfanilamide in a dose of 0.1 Gm. per kilogram by oral administration died in about forty-eight hours (table 4). Of 4 given 0.04 Gm. per kilogram by subcutaneous injection, 2 died in forty-eight hours and 2 survived about seventy-two hours, which indicates a possible slight therapeutic effect of the compound. Four given 0.008 Gm. per

TABLE 4.—*Sulfanilamide in the Treatment of Type I Pneumococcic Meningitis in Rabbits* *

	Administration,† Gm. per Kilogram			
No.	Oral	Subcutaneous	Intracisternal	Results‡
4	0.1	....	.....	All died in 48 hr.
4	...	0.04	.....	2 died in 48 hr.; 2 survived 72 hr.
4	...	....	0.008	All died in from 24 to 48 hr.
4	...	0.04	0.008	1 died in 48 hr.; 3 survived 72 hr.
4	0.1	0.04	0.008	All survived from 72 to 84 hr.
4	Controls.....			All died in from 24 to 48 hr.

* Infected by the intracisternal injection of 0.3 cc. per kilogram of a 1:60 dilution of eighteen hour hormone broth culture.

† Four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation.

‡ All had purulent spinal fluid, with positive cultures.

kilogram by intracisternal injection died in from twenty-four to forty-eight hours. Of 4 given 0.04 Gm. subcutaneously and 0.008 Gm. intracisternally per kilogram, 1 died in forty-eight hours and the remaining 3 survived seventy-two hours. Of 4 given 0.1 Gm. orally, 0.04 Gm. subcutaneously and 0.008 Gm. intracisternally per kilogram, all survived from seventy-two to eighty-four hours.

Of 4 rabbits infected with type II pneumococcus and given 0.1 Gm. of sulfanilamide orally per kilogram of weight, 2 survived four days and 2 five days; their lives were distinctly prolonged as compared with those of 4 untreated controls (table 5). Of 4 given 0.04 Gm. per kilogram by subcutaneous injection, 2 died in about forty-eight hours and the remaining 2 in about four days. Of 4 given 0.008 Gm. per kilogram by intracisternal injection, 3 died in about forty-eight hours and the remaining animal in about four days, the results indicating that the drug administered by this route was apparently without therapeutic effect. Of 4 given 0.04 Gm. subcutaneously and 0.008 Gm. intracisternally per kilogram, 2 died in fifty-four hours, while the remaining 2 survived for about five days. Of 4 given 0.1 Gm. orally, 0.04 Gm. subcutaneously and 0.008 Gm. intracisternally per kilogram, 1 died in seventy-two hours, while the remaining 3 lived for four or five days.

Of 4 rabbits infected with type III pneumococcus and given 0.1 Gm. of sulfanilamide orally per kilogram, 2 died in forty-eight hours and the remaining 2 in about ninety-six hours, the results being simlar to those observed with 4 untreated controls (table 6). Practically the same results were observed with 4 animals given 0.04 Gm. per kilogram by subcutaneous injection, while the intracisternal injection of 0.008 Gm. per kilogram into 4 additional animals was certainly without any evidence of therapeutic effectiveness, since all died in about forty-eight hours.

Likewise, the treatment of 4 animals with 0.04 Gm. administered subcutaneously and 0.008 Gm. administered intracisternally per kilogram was without apparent

TABLE 5.—*Sulfanilamide in the Treatment of Type II Pneumococcic Meningitis in Rabbits* *

	Administration,† Gm. per Kilogram			
No.	Oral	Subcutaneous	Intracisternal	Results‡
4	0.1	....	.....	2 survived 4 days; 2 survived 5 days
4	...	0.04	.....	2 died in 48 hr.; 2 survived 4 days
4	...	....	0.008	3 died in 48 hr.; 1 survived 4 days
4	...	0.04	0.008	2 died in 54 hr.; 2 survived 5 days
4	0.1	0.04	0.008	1 died in 72 hr.; 3 survived 4 or 5 days
4	Controls.....			3 died in 48 hr.; 1 survived 72 hr.

* Infected by the intracisternal injection of 0.3 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth culture.
† Four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation.
‡ All had positive spinal fluid cultures.

TABLE 6.—*Sulfanilamide in the Treatment of Type III Pneumococcic Meningitis in Rabbits* *

	Administration,† Gm. per Kilogram			
No.	Oral	Subcutaneous	Intracisternal	Results‡
4	0.1	....	.....	2 died in 48 hr.; 2 survived 96 hr.
4	...	0.04	.....	2 died in from 48 to 72 hr.; 2 survived 5 days
4	...	....	0.008	All died in 48 hr.
4	...	0.04	0.008	2 died in 48 hr.; 2 survived 4 to 5 days
4	0.1	0.04	0.008	3 survived 72 hr.; 1 survived 6 days
4	Controls.....			2 died in 48 hr.; 2 died in 3 or 4 days

* Infected by the intracisternal injection of 0.3 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth culture.
† Four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation.
‡ All had positive spinal fluid cultures.

benefit, but in 4 additional animals given 0.1 Gm. orally, 0.04 Gm. subcutaneously and 0.008 Gm. intracisternally per kilogram, some therapeutic effect was apparent, since 3 survived for seventy-two hours and 1 for six days, while all 4 untreated controls died from two to four days after infection.

Two untreated monkeys infected intracisternally with type I pneumococci died in from twenty-four to thirty hours of fulminating meningitis and associated septicemia (table 7).

Two given 0.05 Gm. of sulfanilamide orally per kilogram died in about forty-eight hours. But 2 given 0.048 Gm. per kilogram by subcutaneous injection lived for seventy-two and ninety-six hours, respectively, which indicated some possible therapeutic effects of the compound. More conclusive evidence of the therapeutic effectiveness of sulfanilamide was shown in the case of 2 animals given 0.025 Gm.

orally and 0.024 Gm. subcutaneously per kilogram, since 1 survived eighty-four hours and the second about five days.

Type II pneumococci produced fatal meningitis with associated septicemia in an untreated control about twenty-four hours after inoculation. A monkey given 0.1 Gm. of sulfanilamide orally and 0.04 Gm. subcutaneously per kilogram four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation survived for fifty-six hours, while a second given the same dose orally and subcutaneously and two intracisternal injections of 0.008 Gm. per kilogram, twenty-four and forty-eight hours after inoculation, died in forty-eight hours. While the

TABLE 7.—*Sulfanilamide in the Treatment of Type I Pneumococcic Meningitis in Monkeys* *

No.	Administration,† Gm. per Kilogram		Results‡
	Oral	Subcutaneous	
2	0.05	.....	Both died in 48 hr.
2	....	0.048	Both died in from 72 to 96 hr.
2	0.025	0.024	Both survived from 84 to 120 hr.
2	Controls.....		Both died in from 24 to 30 hr.

* Infected by the intracisternal injection of 0.3 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth culture.
† Four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation.
‡ All spinal fluids were purulent, with positive cultures; all cultures of heart blood were positive.

TABLE 8.—*Sulfanilamide in the Treatment of Type II and Type III Pneumococcic Meningitis in Monkeys* *

Type of Infection	Administration, Gm. per Kilogram			Results
	Oral†	Subcutaneous†	Intracisternal‡	
II	0.1	0.04	.....	Died in 56 hr.
II	0.1	0.04	0.008	Died in 48 hr.
III	0.1	0.04	.....	Died in 72 hr.
III	0.1	0.04	0.008	Lived indefinitely
II	Control.....			Died in 24 hr.
III	Control.....			Died in 20 hr.

* Infected by the intracisternal injection of 0.5 cc. per kilogram of a 1:40 dilution of eighteen hour hormone broth culture.
† Four, twenty-four, thirty, fifty-four and seventy-two hours after inoculation.
‡ Twenty-four and forty-eight hours after inoculation.

number of treated animals was too small to permit definite deductions, the results appear to indicate that sulfanilamide prolonged the lives of the 2 treated animals as compared with that of the untreated control (table 8).

Type III pneumococci produced fatal meningitis about twenty hours after inoculation. One animal, given 0.1 Gm. of sulfanilamide orally and 0.04 Gm. subcutaneously per kilogram four, twenty-four, thirty, fifty-four and seventy-two hours after inoculation, lived for seventy-two hours, while a second, given the same dose orally and subcutaneously and 0.008 Gm. per kilogram intracisternally twenty-four and forty-eight hours after inoculation, lived indefinitely. These facts suggest that the compound had a more pronounced effect on type III pneumococci, especially when given intracisternally as well as orally and subcutaneously (table 8).

It would appear, therefore, that sulfanilamide may possess some effectiveness in the treatment of rabbits and monkeys with experimental pneumococcic meningitis and associated septicema, for the lives of some of the treated animals, especially those with type I or type III infection, were prolonged. The evidence of therapeutic effectiveness, however, is not nearly as apparent as that observed in the treatment of meningitis due to beta hemolytic streptococci.

THE MECHANISM OF THE THERAPEUTIC ACTIVITY OF SULFANILAMIDE

The mechanism by which sulfanilamide and its derivatives exert curative activity in bacterial infections is still uncertain.

Domagk [15] has stated that phagocytosis of streptococci by the leukocytes plays an important role in clearing the peritoneal cavity and tissues of mice with experimental peritonitis. He apparently believes that sulfanilamide and its derivatives act on the cocci, either directly or indirectly, in such manner as to render them more susceptible to phagocytosis, and he observed degenerated organisms in the exudates of treated animals. Levaditi and Vaisman [16] soon afterward advanced the hypothesis that capsule formation is retarded, which facilitates phagocytosis.

Colebrook and his colleagues [17] ascribed the curative activity to an increased bacteriostatic and bactericidal activity of the blood, supplemented by that of the tissues of the whole animal. Mellon, Gross and Cooper [18] stated that phagocytosis is no factor in the mechanism of the curative activity, and more recently Gross and his associates [19] questioned that the phagocytic activity of the polymorphonuclear leukocytes and

15. Domagk, G.: Ein Beitrag zur Chemotherapie der baktieriellen Infektionen, Deutsche med. Wchnschr. **61**:250 (Feb. 15) 1935; Untersuchungen über den Wirkungsmechanismus des Prontosil bei der experimentellen Streptokokkeninfektion, Ztschr. f. klin. Med. **132**:775, 1937.

16. Levaditi, C., and Vaisman, A.: L'action préventive du chlorhydrate de 4'sulfamido-2.4-diaminoazobenzène dans l'infection streptococcique expérimentale de la souris, Compt. rend. Soc. de biol. **121**:803, 1936.

17. Colebrook, L., and others: Treatment of Human Puerperal Infections, and of Experimental Infections in Mice, with Prontosil, Lancet **1**:1279 (June 6) 1936. Colebrook, L.; Buttle, G. A. H., and O'Meara, R. A. Q.: Mode of Action of *p*-Aminobenzenesulphonamide and Prontosil in Hemolytic Streptococcal Infections, ibid. **2**:1323 (Dec. 5) 1936.

18. Mellon, R. R.; Gross, P., and Cooper, F. B.: Experimental Studies with Sulfanilamide and with Prontosil in Hemolytic Streptococcus Infections, J. A. M. A. **108**:1858 (May 29) 1937.

19. Gross, P.; Cooper, F. B., and Peebles, M. L.: Effect of Splenectomy on Therapeutic Action of *p*-Amino-Benzenesulfonamide on Mice Infected with Hemolytic Streptococcus, Proc. Soc. Exper. Biol. & Med. **36**:311 (April) 1937.

monocytes is of importance in controlling experimental streptococcic infections in mice treated with sulfanilamide.

Bliss and Long,[20] however, are now of the opinion that sulfanilamide exerts sufficient bacteriostasis in vivo to reduce the rate of multiplication of streptococci and of Clostridium welchii and thus promotes phagocytosis by the leukocytes and disposal of the organisms. Practically the same conclusion has been arrived at by Gay and Clark [21] in the treatment of experimental streptococcic empyema in rabbits. According to their observations sulfanilamide apparently produces sufficient bacteriostasis in vivo to protect the leukocytes in the pleural cavity and to allow the naturally defensive macrophages to accumulate, with the promotion of phagocytosis.

TABLE 9.—*Influence of Sulfanilamide on the Phagocytosis of Streptococci in the Spinal Fluid of Rabbits with Meningitis*

	Untreated Control		Treated Rabbit‡		Treated Rabbit§	
Hours After Infection*	Percentage of Leukocytes, Phagocytic	Phagocytic Index†	Percentage of Leukocytes, Phagocytic	Phagocytic Index	Percentage of Leukocytes, Phagocytic	Phagocytic Index
4	13	0.24	10	0.44	22	0.24
24	10	1.12	35	4.92	10	4.24
48	29	1.24	16	2.76	16	3.80
72	Died	0	Died	0	12	3.44

* Intracisternal injection of 0.3 cc. per kilogram of a 1:30 dilution of eighteen hour hormone broth culture.

† Phagocytic index: average number of cocci per leukocyte.

‡ Given 0.1 Gm. orally and 0.04 Gm. subcutaneously per kilogram, four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation.

§ Given 0.1 Gm. orally and 0.04 Gm. subcutaneously per kilogram, four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation with 0.008 Gm. per kilogram by intracisternal injection at four, twenty-four, forty-eight and seventy-two hour intervals.

In our experiments with sulfanilamide in the treatment of experimental streptococcic meningitis in rabbits and monkeys, there was usually some evidence (especially in monkeys) of increased phagocytosis of the cocci in the purulent spinal fluids by the polymorphonuclear leukocytes.

The results of one experiment with rabbits infected intracisternally with the hemolytic streptococcus are summarized in table 9.

The cisternal fluid of the untreated control four hours after inoculation showed that 13 per cent of the polymorphonuclears had become phagocytic, with an average of 0.24 coccus per leukocyte (designated as the phagocytic index). Twenty-four hours after inoculation 10 per cent of the polymorphonuclears were phagocytic, with an index of 1.12; about forty-eight hours after inoculation 29 per cent of the polymorphonuclears had become phagocytic, with an index of 1.24. In spite of this evidence of phagocytic resistance, the animal died.

20. Bliss, E. A., and Long, P. H.: Observations on the Mode of Action of Sulfanilamide, J. A. M. A. **109**:1524 (Nov. 6) 1937.

21. Gay, F. P., and Clark, A. R.: On the Mode of Action of Sulfanilamide in Experimental Streptococcus Empyema, J. Exper. Med. **66**:535 (Nov.) 1937.

A second rabbit was given 0.1 Gm. of sulfanilamide orally and 0.04 Gm. subcutaneously per kilogram four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation. Cisternal fluid drawn four hours after inoculation, before the first treatment, showed that about 10 per cent of the polymorphonuclears were phagocytic, with an index of 0.44. Twenty-four hours later, after the first treatment but before the second, 35 per cent of the polymorphonuclears were phagocytic, with an index as high as 4.92. Forty-eight hours after inoculation, after the first three treatments, 16 per cent of the polymorphonuclears were phagocytic, with an index of 2.76. The animal died at this time.

A third rabbit was given 0.1 Gm. of sulfanilamide orally and 0.04 Gm. subcutaneously per kilogram four, twenty-four, thirty, forty-eight, fifty-four and seventy-two hours after inoculation and 0.008 Gm. per kilogram at four, twenty-four, forty-eight and seventy-two hour intervals. Four hours later, before the first treatment, 22 per cent of the polymorphonuclear leukocytes were phagocytic, with an index of 0.24. Twenty-four hours later, after the first treatment, 10 per cent of the leukocytes were phagocytic, but with an index of 4.24. Forty-eight hours after infection, after three oral and subcutaneous and two intracisternal treatments, 16 per cent of the polymorphonuclears were phagocytic, with an index of 3.8. Seventy-two hours after infection, after five oral and subcutaneous and three intracisternal treatments, 12 per cent of the polymorphonuclears were phagocytic, with an index of 3.44. This animal survived indefinitely.

In the spinal or cisternal fluids of untreated controls the cocci were almost entirely extracellular, very numerous and in chains. In the case of animals surviving infection, the cocci were numerically distinctly reduced and phagocytosis was definite. We could observe no pleomorphic or degenerative changes in the cocci and no evidence of capsule formation; furthermore, cultures were apparently unchanged in virulence, so far as could be determined by the production of meningitis. It was usually apparent, however, that sulfanilamide reduced the numbers of streptococci and in some manner promoted their phagocytosis. Therefore, we are inclined to agree with Domagk, Bliss and Long, and Gay and Clark that sulfanilamide apparently restrains the growth of beta hemolytic streptococci and thereby facilitates their phagocytosis, probably by reducing production of leukocidins and other antiphagocytic substances or by destruction of toxins. We cannot state how much free and conjugated sulfanilamide entered the cerebrospinal fluid of our animals from oral and subcutaneous injections, as the amounts obtained were required for bacteriologic examinations, but, as shown by Marshall, Emerson and Cutting,[22] the compound when administered orally is to be found in a few hours in the cerebrospinal fluid of the normal dog in a concentration slightly lower than the concentration in the blood, and its passage is likely to be facilitated by the presence of meningitis.

22. Marshall, E. K., Jr.; Emerson, K., Jr., and Cutting, W. C.: Para-Aminobenzenesulfonamide: Absorption and Excretion; Method of Determination in Urine and Blood, J. A. M. A. **108**:953 (March 20) 1937.

METHOD SUGGESTED FOR THE TREATMENT OF STREPTOCOCCIC AND PNEUMOCOCCIC MENINGITIS IN MAN

The encouraging results already reported on the use of sulfanilamide in the treatment of streptococcic meningitis, supplemented by those observed in this experimental study, justify a thorough trial of the drug in the treatment of this highly mortal disease. Much less is to be expected from its use in the treatment of pneumococcic meningitis; nevertheless, it has yielded better results than those observed with other chemical agents [23] and appears to be worthy of clinical trial.

However, it is apparent that reliance is not to be placed on sulfanilamide alone in the treatment of either type. We firmly believe in the advisability and necessity of establishing the best possible drainage of the primary focus of infection and continuous drainage of the cerebrospinal fluid or at least thorough drainage by lumbar puncture every eight to twelve hours.

Sulfanilamide should be administered as soon as meningitis is suspected. Oral administration appears to be about as effective as intramuscular injection, but we believe both routes should be used at least for the first three to five days of treatment. Intraspinal injections do not appear to increase materially the therapeutic effects, but we have some evidence to indicate that administration by this route increases the phagocytosis in animals with experimental streptococcic meningitis.

Sulfanilamide is usually dispensed in tablets carrying 5 grains, or 0.3 Gm., for oral administration. A solution for subcutaneous, intramuscular, intravenous and intraspinal injection is best prepared by dissolving 0.8 Gm. of sulfanilamide powder in 100 cc. of sterile saline solution, brought to the boiling point and cooled to 90 C.; 10 cc. carries 0.08 Gm. of the compound.

During its administration it is our custom to make daily total and differential leukocyte counts and to reduce the dose if there is a reduction in leukocytes thought to be due to depression of the bone marrow by the compound. A careful watch is maintained for the cutaneous manifestations of toxicity, but we do not materially alter the dose because of cyanosis, since this does not appear to be enterogenous, as originally surmised, but is probably due to a by-product in the compound.

In view of the usual fulminating course of streptococcic and pneumococcic meningitis, large doses are required for at least the first three

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23. Kolmer, J. A.: Chemotherapy and Serum Therapy of Pneumococcus and Streptococcus Meningitis: Résumé of Present Status of Treatment of Septic Meningitis, with Recommendation of Method, Arch. Otolaryng. **3**:491 (June) 1926; Pneumococcus and Streptococcus Meningitis: Chemotherapy and Serum Therapy, with Especial Reference to Newer Methods, J. A. M. A. **92**:874 (March 16) 1929; Intracarotid Method of Treatment for Meningitis with Recoveries, ibid. **96**:1358 (April 25) 1931. Kolmer, Rule and Madden.[13]

to five days of treatment. The amounts advised for oral or intramuscular administration alone or in combination are summarized in table 10.

Since sulfanilamide has been found by Schwentker, Gelman and Long [24] and others to be of value in the treatment of meningococcic meningitis, the same dose may be employed, although it appears advisable to administer antimeningococcus serum intraspinally at the same time in the treatment of this type of meningitis.

If intraspinal injections of sulfanilamide are given in addition to oral or intramuscular administrations in the treatment of streptococcic and pneumococcic meningitis, from 5 to 20 cc. of the 0.8 per cent solution may be administered after drainage of the spinal fluid every twelve hours, as shown in table 10; these amounts do not materially increase the total dose per twenty-four hours.

TABLE 10.—*Suggested Dose of Sulfanilamide in the Treatment of Meningitis*

Approximate Weight, Pounds	Oral Alone, Tablets		Intramuscular Alone, Cc. of 0.8% Solution		Combined Oral and Intramuscular			Intraspinal, Every 12 Hr., 0.8% Solution
	Every 6 Hr.	Total, 24 Hr.	Every 6 Hr.	Total, 24 Hr.	Oral, Every 6 Hr.	Intramuscular, Every 6 Hr.	Total, 24 Hr., Gm.	
20	½	2 (0.6 Gm.)	12	50 (0.4 Gm.)	½ tablet	10 cc.	0.68	5 cc. (0.04 Gm.)
50	2	8 (2.5 Gm.)	50	200 (1.6 Gm.)	2 tablets	25 cc.	2.70	10 cc. (0.08 Gm.)
100	3	12 (3.5 Gm.)	75	300 (2.4 Gm.)	3 tablets	50 cc.	4.00	15 cc. (0.12 Gm.)
Adults	4	16 (5.0 Gm.)	100	400 (3.2 Gm.)	4 tablets	50 cc.	5.40	20 cc. (0.16 Gm.)

Under favorable conditions the dose may be reduced after from three to five days, but oral administration is advised for at least two or three weeks as a safeguard against a recrudescence of the infection.

SUMMARY AND CONCLUSIONS

In rabbits the intracisternal inoculation of virulent beta hemolytic streptococci produced acute fulminating meningitis, with associated septicemia, which was fatal in from eighteen to forty-eight hours. The treatment of 46 rabbits with prontosil solution and sulfanilamide, begun four hours after inoculation, resulted in the recovery of 9 and the prolongation of the lives of most of the remaining animals.

The intracisternal inoculation of monkeys with streptococci produced a meningitis which was fatal in about seventy-two hours but was unaccompanied by septicemia. Treatment of 12 animals with sulfanil-

24. Schwentker, F. F.; Gelman, S., and Long, P. H.: Treatment of Meningococcic Meningitis with Sulfanilamide: Preliminary Report, J. A. M. A. **108**:1407 (April 24) 1937.

amide, begun twenty-four hours after inoculation, resulted in the recovery of 7.

The intracisternal inoculation of rabbits with virulent types I, II and III pneumococci produced acute meningitis, with associated septicemia, which was usually fatal in from two to four days. Treatment of 60 animals with sulfanilamide, begun four hours after inoculation, did not result in the recovery of any but apparently prolonged the lives of 24 for various periods.

The intracisternal inoculation of monkeys with virulent types I, II and III pneumococci produced fatal meningitis, with associated septicemia, which was fatal in from twenty-four to thirty hours. Treatment of 10 animals with sulfanilamide, begun twenty-four hours after inoculation, resulted in the recovery of 1 (type III) and apparently prolonged the lives of 7 others.

Intraspinal injections of sulfanilamide in doses of 0.008 Gm. per kilogram (1 cc. of 0.8 per cent solution) were well borne but did not produce as favorable results as the oral or the subcutaneous administrations of the compound. The best results were observed with a combination of oral administration and subcutaneous and intraspinal injections, although a combination of oral administration and subcutaneous injections appeared to give about as good results as a combination of the three methods.

The therapeutic activity of sulfanilamide in the treatment of streptococcic meningitis appears to depend on the promotion of phagocytosis of streptococci, which is probably the result of some bacteriostatic effect resulting in the production of less antiphagocytic substance.

The results indicate that maximum doses of sulfanilamide administered as early as possible may be therapeutically helpful in the treatment of streptococcic meningitis in human beings and worth trying in the treatment of pneumococcic meningitis. A scale of suggested doses to be administered by various routes is submitted.

STUDIES IN THE MECHANISM OF THE ACTION OF SULFANILAMIDE

II. SULFANILAMIDE IN THE TREATMENT OF EXPERIMENTAL STREPTOCOCCIC MENINGITIS

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Since the introduction of sulfanilamide (paraaminobenzenesulfonamide), many reports [1] have appeared concerning the successful treatment of meningitis due to Streptococcus haemolyticus in man. This is of considerable importance, since the mortality rate for all types of streptococcic meningitis has been estimated by Gray [2] at 97 per cent with means of treatment other than sulfanilamide. Any therapy that gives promise of materially lowering this mortality rate is indeed welcome, even if it is accompanied by minor untoward effects, such as those indicated by recent reports on toxic manifestations due to sulfanilamide.[3]

A review of the literature reveals that in most of the animal experiments in which sulfanilamide was used to treat infection due to Str. haemolyticus, the infection was confined to tissues other than the meninges;[4] i. e., subcutaneous infection, intraperitoneal infection or

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From the Laboratory of Surgical Bacteriology, Harrison Department of Surgical Research, University of Pennsylvania School of Medicine.

1. Schwentker, F. F., and others: Bull. Johns Hopkins Hosp. **60**:297, 1937. Caussé, Loiseau and Gisselbrecht: Ann. d'oto-laryng., February 1936, p. 194. Carey, B. W., Jr.: J. Pediat. **11**:202, 1937. Trachsler, W. H.; Frauenberger, G. S.; Wagner, C., and Mitchell, A. G.: ibid. **11**:248, 1937. Anderson, E. D.: Hemolytic Streptococcus Meningitis: Report of a Case with Recovery After the Use of Prontosil and Sulfanilamide, J. A. M. A. **108**:1591 (May 8) 1937. Arnold, J. G., Jr.: Ann. Int. Med. **10**:1198, 1937.

2. Gray, H. J.: Streptococcic Meningitis: Report of a Case with Recovery, J. A. M. A. **105**:92 (July 13) 1935.

3. Discombe, G.: Lancet **1**:626, 1937. Kohn, S. E.: Acute Hemolytic Anemia During Treatment with Sulfanilamide, J. A. M. A. **109**:1005 (Sept. 25) 1937. Bucy, P. C.: Toxic Optic Neuritis Resulting from Sulfanilamide, ibid. **109**:1007 (Sept. 25) 1937. Young, C. J.: Brit. M. J. **2**:105, 1937.

4. Domagk, G.: Deutsche med. Wchnschr. **61**:250, 1935. Long, P. H., and Bliss, E. A.: Para-Amino-Benzene-Sulfonamide and Its Derivatives: Experimental and Clinical Observations on Their Use in the Treatment of Infections Due to Beta Hemolytic Streptococci, J. A. M. A. **108**:32 (Jan. 2) 1937. Tréfouël, J.; Tréfouël, J. (Mme.); Nitti, F., and Bovet, D.: Compt. rend. Soc. de biol. **120**: 756, 1935. Raiziss, G. W.; Severac, M., and Moetsch, J. C.: J. Chemotherapy **14**:1, 1937.

septicemia was treated. We are not aware of any reports of experiments carried out on animals to test the effect of sulfanilamide on meningitis due to the hemolytic streptococcus. Moreover, there have been only meager reports of the treatment with sulfanilamide of experimental infection elsewhere than in the serous cavities, and it seemed possible that chemotherapeutic results might vary under different anatomic conditions.

We are reporting animal experiments in which meningitis due to Str. haemolyticus, similar to that occurring in man, was produced under controlled conditions and treated with sulfanilamide. Normal adult white rats (Mus norvegicus albinus) of both sexes, weighing about 200 Gm., were used because of their availability and because of the practical difficulties of handling large groups of rabbits or similar animals. Some doubt arose in the beginning as to the suitability of the rat for these experiments. Judging from some of our preliminary experiments and those of others,[5] the white rat possesses moderate resistance to infection of the meninges due to Str. haemolyticus. According to Zinsser,[6] however, the rat is more susceptible to invasive streptococcic infection than the mouse or the rabbit, and the natural immunity which it does possess is more antitoxic than antibacterial. Since in our experiments we were determining the effect of sulfanilamide in promoting resistance to an invasive type of lesion, there seemed to be no valid fundamental objection to employing the rat as the experimental animal.

Various technics were tried. The method of cisternal puncture as practiced by Griffith, Jeffers and Lindauer,[5] followed by the introduction of a streptococcus culture through the needle, was used at first. This, however, did not result in the uniform production of typical lesions, apparently owing to the lack of a protected focus to act as a propagating nidus, such as is usually present with human otogenic infection. The results of these experiments are presented in table 1.

It was finally found that the most satisfactory method of producing the disease was as follows:

Sterile precautions being taken, a small incision was made over the dorsum of the head while the animal was under ether anesthesia, and a simple opening in the calvarium, about 2 mm. in diameter, was bored with the point of a scalpel by rotating the scalpel on its axis until it had worked its way through to the dura mater. The usual site for the opening was lateral to the midline, just posterior to the frontoparietal suture, and the longitudinal and transverse sinuses were thus avoided. More constant results were produced with inoculations when blood agar was introduced before the culture. This was done by plugging the opening with a small piece of solid agar medium, rather than by injecting melted

5. Griffith, J. Q., Jr.; Jeffers, W. A., and Lindauer, M. A.: Am. J. Physiol. **113**:285, 1935.

6. Zinsser, H., and Bayne-Jones, S.: Textbook of Bacteriology, New York, D. Appleton and Company, 1928, p. 330.

agar, as tried at first, since even sterile melted agar was found to produce death from mechanical effects. The inoculum of streptococci was injected through the agar into and beneath the dura, infecting the dura, the subdural space and the blood agar. The wound in the skin was closed with interrupted silk sutures and the wound painted with iodine.

The strain of hemolytic streptococci used was "1896M," which had been lyophilized after mouse passage about ten months previously.[7] The culture was grown in meat infusion broth for eighteen hours in the incubator at 37 C. A drop of this culture was transferred to blood broth, incubated at 37 C. for eighteen hours and then kept in the ice box at 4 C. In preparing the culture for inoculation a drop of this ice box culture was placed in 5 cc. of neopeptone water containing 5 per cent of horse serum[8] and incubated for approximately eighteen hours. It

TABLE 1.—*Preliminary Experiments in the Preparation of the Final Technic*

Method of Injection	Culture Medium	Str. Haemolyticus	Quantity, Cu. Mm.	Dilution	No. of Rats	Died*	Postmortem Observations
Intracisternal	Neopeptone water	Sterile	50	.....	5	0	
Intracisternal	Neopeptone water	C203M	50	1:10	2	1	
		C203M	50	1:1,000	3	1	
		C203M	50	1:100,000	2	0	
Intracisternal	Neopeptone water	1896M	50	Undiluted	1	1	Streptococcic meningitis
		1896M	50	1:100	2	0	
		1896M	50	1:10,000	2	0	
Subdural, through trephine	Neopeptone water	1896M	150	Undiluted	2	2	
Subdural, through trephine	Melted blood agar	Sterile	100	.....	2	1	Injury to the brain
Extradural, at trephine	Melted blood agar	Sterile	50	.....	3	0	
Subdural, through trephine	Neopeptone water	1896M	100	1:10,000	3	1	Streptococcic meningitis
Subdural, through trephine	Neopeptone water	1896M	100	1:10,000	3	2	Streptococcic meningitis; subcutaneous streptococcic abscess

* Died within ten days.

was then diluted to 1:100 with serum-free neopeptone water, and about 75 cu. mm. was injected through a 24 gage hypodermic needle, a tuberculin syringe being used.

Sulfanilamide was administered for five days by means of a no. 10 French catheter passed into the stomach. It was given in the form of a freshly prepared 10 per cent emulsion in 1.6 per cent acacia. Marshall and his collaborators[9] demonstrated that sulfanilamide is present in the spinal fluid in a concentration only slightly lower than that in which it is

7. Dr. Stuart Mudd, of the Department of Bacteriology, supplied this culture.

8. Ward, H. K., and Lyons, C.: J. Exper. Med. **61**:517, 1935.

9. Marshall, E. K.; Emerson, K., Jr., and Cutting, W. C.: Para-Aminobenzenesulfonamide: Absorption and Excretion; Method of Determination in Urine and Blood, J. A. M. A. **108**:953 (March 20) 1937.

present in the blood. We might have given sulfanilamide intrathecally but wished to check on the efficacy of gastric administration alone, believing that the level in the spinal fluid, per se, was not so important as the general level in the blood and lymph. In our later experiments the drug was administered more than once daily, as it has been shown by the same authors that sulfanilamide is excreted rather rapidly by the kidneys, and the maintenance of a more constant high level in the blood, which is obtained when divided doses are used, is desirable. Admin-

TABLE 2.—*Sulfanilamide Given to Treated Animals in Single Daily Dose*

	No. of Rat	Day of Death	Blood		Meninges (Brain)			Histologic Examination
			Broth	Agar	Smear	Broth	Blood Agar	
Sulfanilamide: 150 mg. daily for five and one-half days (one dose by mouth); first dose one hour after operation	10	2d	0	0	+	+	+	Localized surface exudate on cortex; many organisms present but chiefly surrounded by exudate; many shadows of dead cocci
	20	2d	0	0	+	+	+	Slight inflammatory reaction; rare cocci on surface; no invasion
	11	3d	0	0	+	+	+	Diffuse surface exudate on meninges; heavy infiltration of round cells; cocci limited to areas of exudate; no invasion
	12	Survived						
Controls (no drug)	2	1st	+	+	+	+	+	Extensive arachnoidal involvement; exudate chiefly polymorphonuclear; large numbers of free cocci
	3	2d	+	+	+	+	+	Diffuse spread; exudate polymorphonuclear; large numbers of free cocci on surface and invading cortex
	21	2d	+	+	+	+	+	Diffuse invasive lesion; large masses of free cocci, with little cellular exudate
	22	2d	+	+	+	+	+	Diffuse involvement of surface; no cortical invasion; organisms few
	1	4th	+	+	+	+	+	Moderately diffuse exudate, with invasion and necrosis of cortex; cocci seen beyond zones of exudate and in huge clumps

istration of the drug in smaller amounts but at more frequent intervals would perhaps have been even better.

Almost all the rats, both of the control and of the treated groups, showed at some time definite signs of meningismus, manifested in a stiff arching of the back and spastic hyperextension of the hindlegs. Those that died often had convulsive seizures which caused spasm of the respiratory muscles followed by death from asphyxia. Death was usually preceded by coma lasting for a few hours, during which the rat lay on its side.

As soon as feasible after death, autopsy was performed under strictly aseptic precautions.

The actual cautery was used to burn through the skin, subcutaneous tissues and thorax, so as to expose the heart. A freshly boiled hypodermic needle connected with a sterile tuberculin syringe was then inserted into the heart to withdraw heart blood. This was implanted into a culture tube of meat infusion broth and incubated. If visible growth occurred after eighteen hours of incubation, a loopful was streaked on blood agar. At the same time a smear was made. The blood agar was examined the following day, after incubation, for typical colonies of streptococci with hemolysis.

TABLE 3.—*Sulfanilamide Given to Treated Animals in Two Daily Doses*

	No. of Rat	Day of Death	Blood		Meninges (Brain)			Histologic Examination
			Broth	Agar	Smear	Broth	Blood Agar	
Sulfanilamide: 150 mg. in first dose, immediately after operation; then 75 mg. twice daily for 5 days	1	8th	+	+	+	+	+	Well localized abscess; meninges thickened; many phagocytosed and pyknotic cocci; no diffuse lesion; cord uninvolved
	2	9th	0	0	0	+	+	Slight exudate; spinal meninges involved; a few phagocytosed cocci on meninges of brain; many free cocci in exudate from cord
	3	Survived						
	4	Survived						
	10	Survived						
	11	Survived						
	12	11th	0	0	+	+	+	Extensive surface exudate; few organisms confined to zone of exudate, greatly outnumbered by phagocytes
Controls (no drug)	13	2d	+	0	+	+	+	Widespread exudate, involving cord; great masses of cocci, outnumbering phagocytes
	30	2d	+	+	+	+	+	Extensive infection, with invasion; masses of cocci; no control by phagocytes; cord involved
	21	3d	+	+	+	+	+	Extensive surface exudate; cord involved; phagocytes greatly outnumbered by masses of cocci
	20	3d	+	+	0	+	0	Slight surface exudate; cord not involved; only a few intracellular cocci seen, none free
	22	3d	+	+	0	0	0	Abscess, with extensive necrosis and invasion; huge masses of cocci, outnumbering phagocytes
	31	3d	0	0	0	0	0	Small amount of exudate; few faintly defined intracellular cocci
	40	4th	0	0	0	0	0	Exudate only on surface; no organisms seen

The skull also was exposed by the use of the actual cautery. It was then opened with a small trephine, 6 mm. in diameter, through the middle of the parietal bone on the side opposite the original opening, and a fresh area of the meninges was exposed. A small piece of meningeal tissue was cut off some distance from the point of inoculation and placed in a culture tube of meat infusion broth. It was examined by smear after eighteen hours of incubation, then plated on blood agar and after another eighteen hours of incubation examined for characteristic colonies and hemolysis. A smear was made direct from the meninges, stained with methylene blue and examined; a block of tissue, including a portion of the

meninges, was taken for histologic section, fixed in Zenker's solution and stained by Giemsa's method to demonstrate bacteria. Similar sections were also made of the meninges of the lumbar portion of the spinal cord. These sections were studied for information as to type of exudate, presence or absence of invasion, activity of phagocytosis and morphologic change in the organisms under the influence of sulfanilamide.

TABLE 4.—*Repetition of Experiment Summarized in Table 3*

	No. of Rat	Day of Death	Blood		Meninges			Histologic Examination
			Broth	Agar	Smear	Broth	Blood Agar	
Sulfanilamide: 150 mg. in first dose, immediately after operation; then 75 mg. twice daily for five days	20	3d*	0	0	0	+	+	Surface exudate; no invasion; cord not involved; no organisms
	30	3d*	0	0	..	0	0	Surface exudate; no invasion; cord not involved; rare cocci, mostly pyknotic
	14	3d	0	0	+	+	+	Surface exudate; no invasion; cord involved; few scattered free cocci, large size
	13	4th	0	0	+	0	0	Well localized surface exudate, with abscess; organisms limited to abscess
	40	6th	0	0	0	0	0	Moderate exudate; no organisms
	21	Survived						
	23	Survived						
	22	Survived						
	31	Survived						
Controls (no drug)	10	1st	+	+	+	+	+	Little exudate; marked invasion of cortex; cord involved; great masses of cocci; phagocytes outnumbered
	11	1st	+	+	+	+	+	Thick exudate; some cortical invasion; cord involved; many free organisms within and beyond exudate
	12	2d	+	+	+	+	+	Meninges thickened, with heavy exudate; cortex invaded by free cocci in huge numbers; cord involved
	1	2d	0	0	+	+	+	Scanty exudate; rare free cocci
	2	2d	+	+	+	+	+	Scanty exudate but marked invasion; cord involved; great masses of cocci
	3	2d	0	0	+	+	+	Marked invasion; heavy exudate; masses of cocci; cord involved
	4	2d	+	+	+	+	+	Marked invasion; heavy exudate; masses of cocci; cord involved

* Killed on third day.

ANALYSIS OF DATA

In order to evaluate the therapeutic agent employed it seemed important not only to consider the statistical incidence of survival but to study the effects which the drug might have on the pathologic course of the disease. The data will be considered in sequence and an attempt made to assess their significance not only in relation to the meningeal lesion but with regard to the mode of action of sulfanilamide.

SURVIVAL

For the small group given sulfanilamide in single daily doses (table 2), the survival rate was only 25 per cent and the average length of life of those that died was two and a third days (table 5). The latter figure is about the same as that for the control group, suggesting that whatever modifications in the character of the disease the drug had effected were not of such a nature as to prolong life. For none of the treated animals, however, was a postmortem culture of the heart blood positive, and none showed histologic evidence of an invasive lesion.

When the same total amount of sulfanilamide was given in divided doses, a definite increase in the protective influence of the drug was

TABLE 5.—*Summary of Experiments with Meningitis in Rats: Survival*

	Number	Survived	Death in Average Days
Control animals	19	0	2.26
Treated animals	18	9 (50%)	5.33
Sulfanilamide once daily	4	1 (25%)	2.33
Sulfanilamide twice daily	14	8 (57%)	6.83

TABLE 6.—*Summary of Experiments with Meningitis in Rats: Septicemia (Postmortem Culture)*

	Number	Positive Blood Culture
Control animals	19	15 (80%)
Treated animals	9	1 (11%)*

* This rat died on ninth day, four days after administration of the drug was stopped.

shown, with the incidence of survival increasing to 57 per cent. The average duration of life of the animals which did not survive was six and eighty-three hundredths days, which was longer than the period of administration of sulfanilamide. These facts conform to the general clinical experience that repeated doses at short intervals, preferably every four to six hours, yield the best results.[9]

In addition to the influence of sulfanilamide on mortality, there was evident a clinical alteration in the character of the disease in the treated animals, even in those which finally died. On the third day the rats receiving the drug twice daily became more lively and when released from the cage ran around actively on the table, whereas by that time most of the control animals were dead. Some of the treated rats remained perfectly normal in behavior throughout the experimental period. Surviving animals were watched for two months, and none died of delayed infection.

SEPTICEMIA

Table 6 shows a striking difference between the two groups in the incidence of positive cultures of heart blood made post mortem. The only treated animal whose culture was positive died on the ninth day, four days after treatment with sulfanilamide was stopped. Presumably

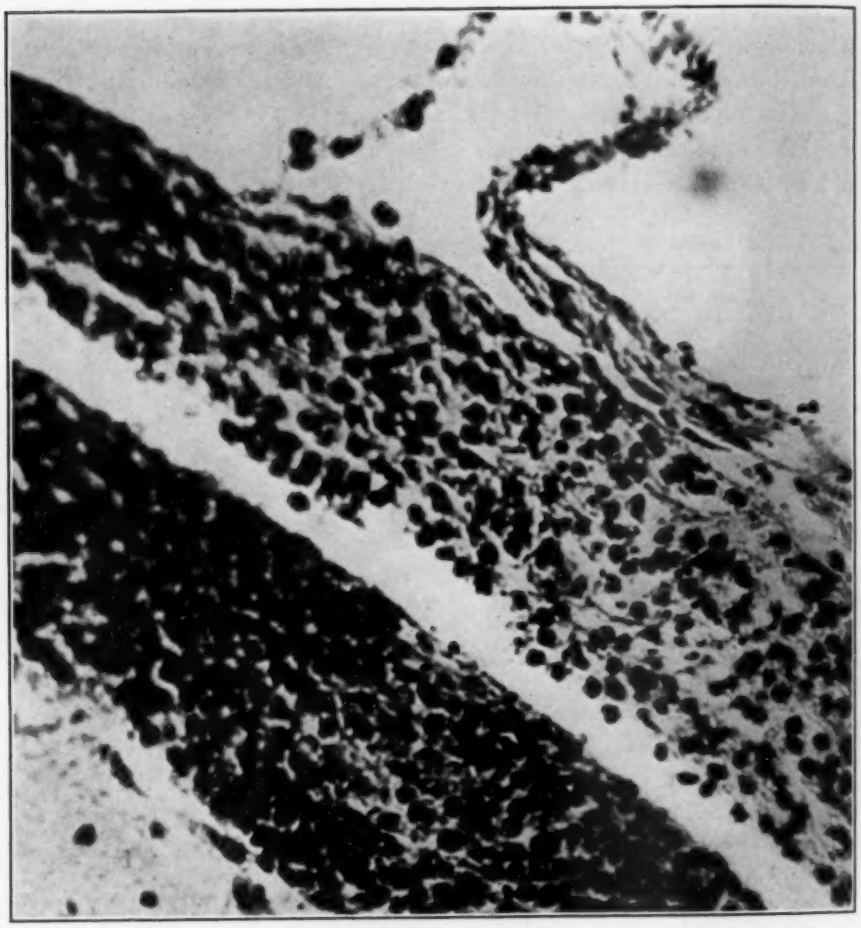

Fig. 1.—Diffuse meningitis, with invasion of the cortex, in a control animal which died after twenty-four hours (× 470).

this rat continued to harbor living streptococci, which were afforded the opportunity to invade the blood stream after the restraining effect of sulfanilamide was withdrawn. Histologically there was no evidence of active meningitis, but the brain showed a well localized cortical abscess (fig. 6).

HISTOLOGIC EXAMINATION

In classifying the pathologic changes we selected three main criteria as being significant, namely, the relative numbers of free cocci in the exudates, the presence or absence of invasion of tissue and the presence

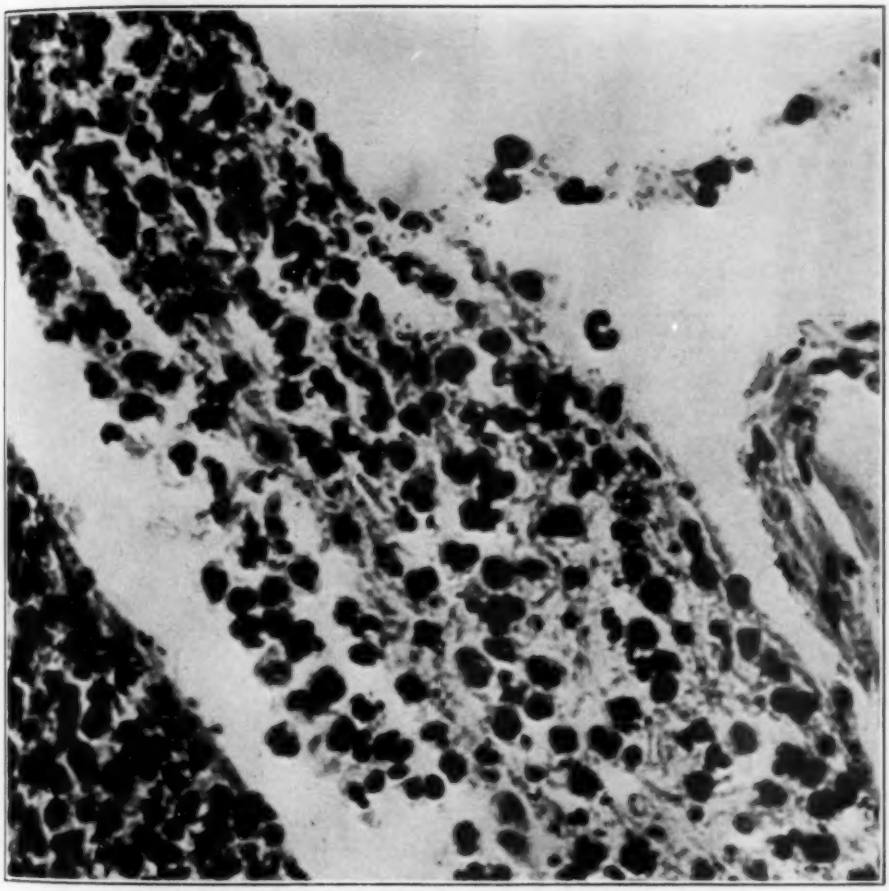

Fig. 2.—Free cocci in a peripheral strand of the dura mater in a control animal which died after twenty-four hours.

or absence of involvement of the spinal meninges. Figures 1 to 8 illustrate the histologic data.

Numbers of Free Cocci.—As shown in table 7, there was a tendency toward unrestricted growth of the streptococci in the exudates and tissues of control animals, 74 per cent of the animals showing large numbers of organisms, arranged as a rule in great masses (fig. 7) or

else singly and in short chains. When invasion by streptococci had extended beyond the limits of the inflammatory exudate, the latter arrangement was the rule. In contrast to this picture, the exudates of treated animals were relatively free from cocci, and when the organisms were present in large numbers they were either confined to a localized abscess or accompanied by profuse infiltration of phagocytes (fig. 7 and 8). There was no striking increase in phagocytosis in the treated animals. It was rather suggested that the streptococci were not capable of multiplying in unrestricted fashion in the treated animals as they

TABLE 7.—*Histologic Examination: Free Streptococci in Surface Exudate or Tissue*

	Large Numbers	Moderate Numbers or Few	None
Control animals	14 (74%)	2 (10%)	3 (16%)
Treated animals	4 (36%)*	4 (36%)	3 (28%)

* In 2 the organisms were confined to an abscess, and in 2 the surrounding exudate was rich in phagocytes.

TABLE 8.—*Histologic Examination: Invasion of the Cortex*

	Number	Incidence
Control animals	19	12 (63%)
Treated animals	11	0

TABLE 9.—*Histologic Examination: Involvement of Spinal Meninges*

	Number Examined	Incidence of Involvement
Control animals	14	8 (57%)
Treated animals	8	2 (25%)*

* Died on third and ninth days.

seemed to do in the controls. There were no striking morphologic differences between the organisms in the two groups, but in the treated group the individual cocci were larger and tended to form slightly longer chains.

Invasion.—By the term invasion we imply the presence of free cocci in tissues not yet infiltrated by the inflammatory exudate. In the belief that streptococcic invasion occurs under conditions which permit relatively unrestrained multiplication and diffusion of the cocci, we include these observations as being of significance. As shown in table 8, this type of invasion was observed in 63 per cent of the control animals and in none of the treated animals. This could be attributable either to slower multiplication of cocci in the treated than in the control

animals or to more active phagocytosis in the former than in the latter. In the light of the observations mentioned in the previous paragraph, it seemed to us that absence of invasion in treated animals could be more satisfactorily explained on the basis of marked bacteriostasis induced by the sulfanilamide.

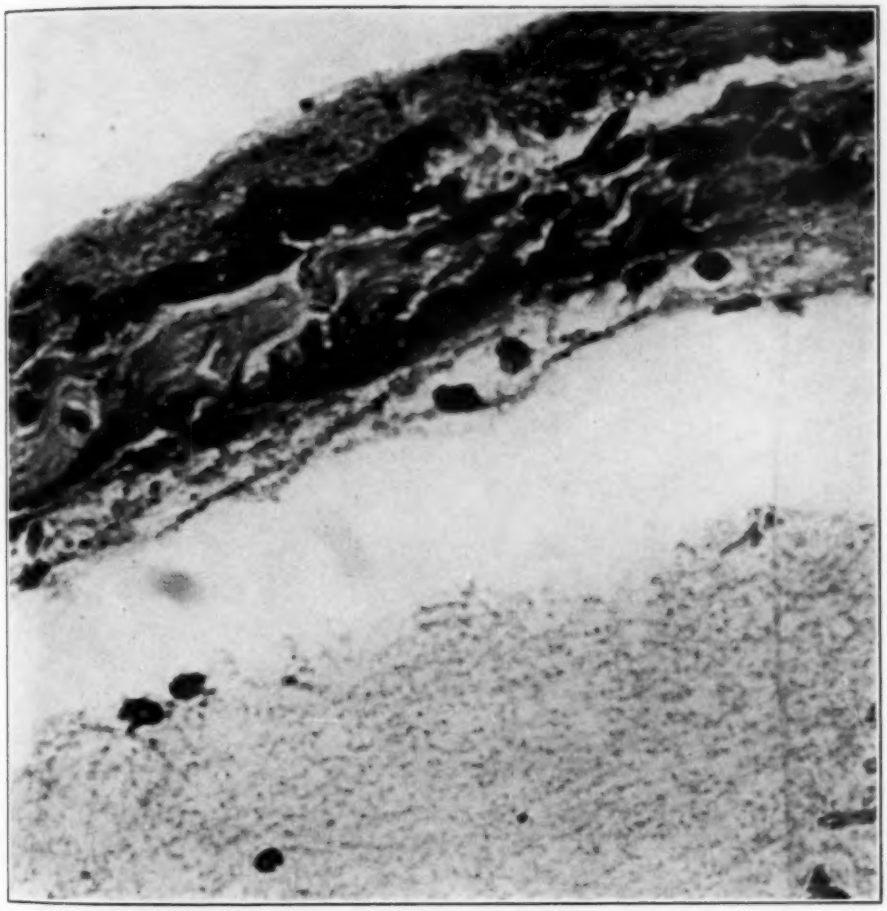

Fig. 3.—Thickened meninges, acellular exudate and rare free cocci in an animal which was given sulfanilamide once daily and died after forty-eight hours (× 810).

Involvement of the Spinal Meninges.—In view of the fact that the organisms were introduced through the cranium, it is noteworthy that 57 per cent of the control animals showed involvement of the spinal meninges while in only 2, or 25 per cent, of the group treated with sulfanilamide did such involvement occur. One treated animal died on

the ninth day, after having shown evidence of severe meningeal irritation on the fourth day, which then temporarily subsided, recurring the day before death. Presumably, the infection remained under partial control until withdrawal of sulfanilamide permitted further extension to take place.

Fig. 4.—Exudate within a blood vessel in an animal which was given sulfanilamide once daily and died after forty-eight hours; relative absence of tissue reaction in meninges and cortex (× 810).

COMMENT

Fundamentally streptococcic meningitis is cellulitis of a highly vascular structure, the meninges. It is to some extent meningoencephalitis. It seems to us that the chemotherapeutic approach to such a lesion is logically directed toward providing the meninges and the

underlying tissues with an environment unfavorable for their invasion by the streptococci. Sulfanilamide administered by mouth seems to accomplish this objective. If one accepts the reasonable belief that it is the organisms in the inflammatory tissue "frontier" and in the blood vessels which are participating in, and responsible for, further spread

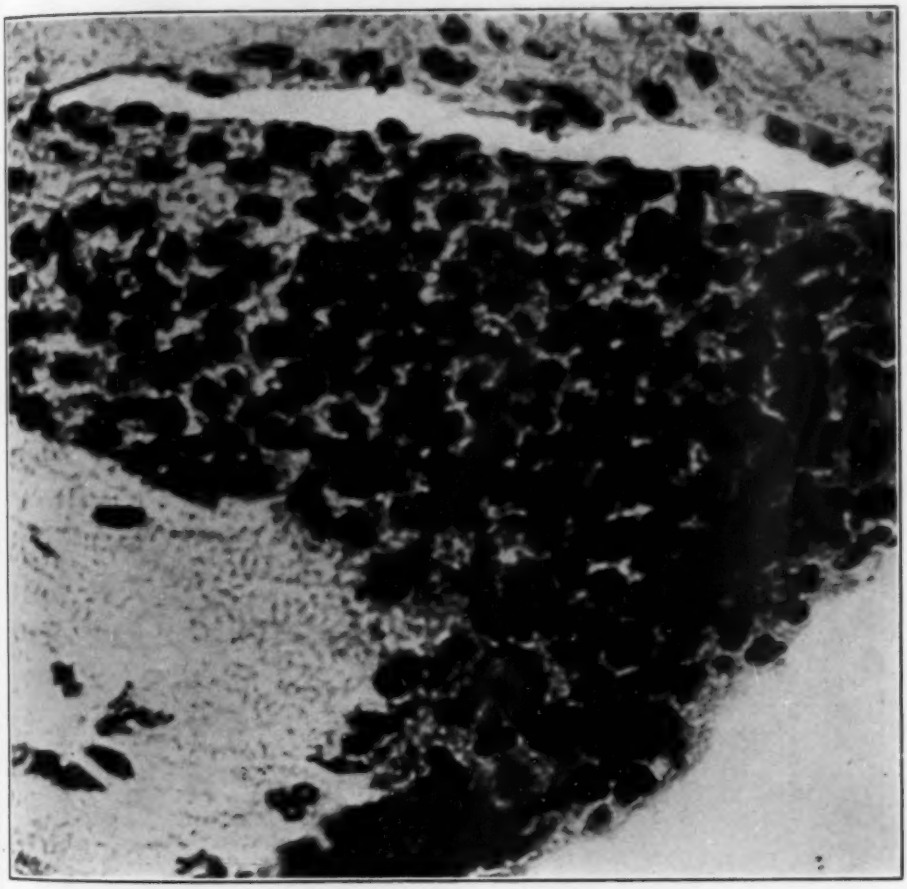

Fig. 5.—Invasion of the cortex in a control animal which died after twenty-six hours (× 810).

of the infection, one will not put much faith in an effort to apply the chemical through the intrathecal route. Our experimental results indicate that the oral administration of the drug is effective in modifying the disease.

In treating this type of lesion the important factor is to attain an adequate level of sulfanilamide in the blood. Marshall [9] has shown

that the drug is present in the spinal fluid in a concentration slightly below that found in the blood. Furthermore, the relative insolubility of sulfanilamide makes it possible to give only trivial amounts intrathecally. We therefore would emphasize that the logical as well as the

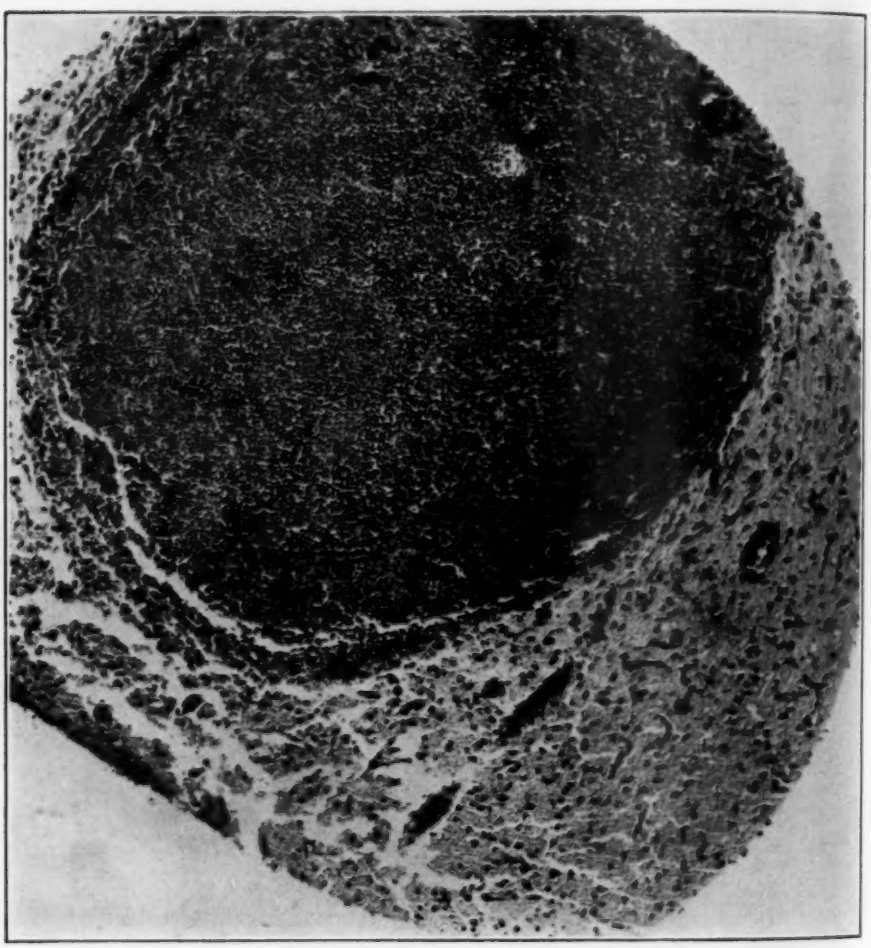

Fig. 6.—Localized abscess in an animal which was given sulfanilamide twice daily and died on the eighth day (× 110).

practical methods of administration of sulfanilamide in the treatment of streptococcic meningitis are through the oral and the subcutaneous route.

If sulfanilamide can be given coincidentally with the introduction of virulent organisms into the meninges and produce significant alterations

in the course of the disease, it might reasonably be expected to be even more effective in preventing an infection from spreading to this region. It is therefore further suggested that the practice of giving sulfanilamide to patients with streptococcic otitis media and mastoiditis is to be strongly recommended, particularly when extensive surgical interven-

Fig. 7.—Great masses of free cocci and few leukocytes in a control animal which died after forty hours (× 1,400).

tion seems likely to be required. Whether or not the initial lesion is "cured" by sulfanilamide is of secondary importance if further invasion from that lesion is prevented.

It is of course impossible to draw definitive conclusions regarding the mode of action of sulfanilamide in vivo from such experiments as

those now being reported. However, it is our impression that the drug acts not by promoting phagocytosis but rather by restricting the multiplication of the cocci and preventing invasion of the tissues and the blood stream. Other experiments bearing on this point are to be reported by one of us (J. S. L.) in a subsequent paper.

Fig. 8.—Inflammatory exudate without any cocci in an animal which was given sulfanilamide twice daily and killed after forty-eight hours (× 2,620).

SUMMARY AND CONCLUSIONS

1. Sulfanilamide administered by mouth is a potent therapeutic agent against meningitis due to Str. haemolyticus in the white rat. Even when death is not prevented, the character of the lesion is modified.

2. Sulfanilamide must be given at least twice daily to be effective in influencing mortality, and presumably more frequent doses would be ideal.

3. It does not appear to be necessary to give the drug intrathecally, at least under experimental conditions.

4. Sulfanilamide, even when given in doses insufficient to prevent death from meningitis due to Str. haemolyticus, provides the blood with sufficient antibacterial power to resist invasion of the blood stream.

5. The effect of sulfanilamide seems to be the provision of a tissue environment in which the cocci are unable to multiply freely and to invade adjacent tissues and the blood stream.

RETROPHARYNGEAL AND CERVICAL EMPHYSEMA

REPORT OF A CASE

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SAN FRANCISCO

Subcutaneous emphysema is usually due to distention of the subcutaneous areolar tissue with air. Diagnosis of the condition and determination of its cause are not as a rule difficult. Its commonest starting place is in the thorax, particularly where there has been injury to the tissue of the lung, as by a broken rib, a stab with a knife, a bullet wound or rupture of an alveolus by excessive coughing (as in whooping cough or bronchitis) or in an operative procedure (such as exploratory needling of the chest). The air spreads rapidly and may extend over the greater part of the trunk in a short time, disappearing in the course of a few days. It may act similarly after a tracheotomy. The face may sometimes be almost suddenly involved unilaterally by the escape of air into the subcutaneous tissues from the upper part of the nose, after violent sneezing or energetic blowing of the nose.

The case here reported is of interest because of the presentation of an unusual problem of differential diagnosis, in which the following clinical entities were considered: retropharyngeal abscess and cervical cellulitis, gas bacillus infection, and emphysema. Immediate determination of the cause appeared paramount, since appropriate treatment apparently hinged on the proper diagnosis.

REPORT OF CASE

Present History.—A white man aged 29 was celebrating New Year's Eve by blowing on a tin horn, when the horn was suddenly thrust down his throat. He lost consciousness for a short while, and later there was some hemorrhage from his mouth. Slight bleeding continued, and on Jan. 2, 1938, the left side of his neck below the angle of the jaw began to swell. Rest in bed and hot compresses were ordered by his physician, but his throat internally and his neck externally continued to swell to such an extent as to cause great discomfort and to render respiration and swallowing markedly difficult.

Past History.—The patient had had measles, mumps, pertussis, scarlet fever and influenza in childhood. He had contracted malaria in Louisiana in 1933, and it had recurred in 1934. He had had bronchial asthma as long as he could remember.

Examination.—On the afternoon of January 3 he was hospitalized. I saw him the following morning in consultation with his physician, Dr. Frank M. Close.

The accompanying photographs do not show to great advantage the marked swelling at the angle of the left mandible and in the left posterior cervical triangle which this patient presented. There was no superficial crepitation, but the swelling was soft and only slightly tender. His head was held rigidly, somewhat to the right, and he could open his mouth only a small distance. The soft palate on the left was swollen and edematous to such an extent as to push the uvula (only slightly enlarged) over to the right tonsil. There was a small grayish necrotic spot in the soft palate, just in the left supratonsillar area. The left tonsil could not be brought into view. The entire posterior pharyngeal wall seemed swollen and bulged forward, although there appeared to be no local area of pointing. Ears and nose were normal, and other physical findings were essentially negative. There was some difficulty in respiration, associated with tightness in the muscles of the throat and dysphagia, but no alteration of the voice.

Laboratory Data.—The patient's temperature was 38.5 C. (101.3 F.); his pulse rate, 102, and his respiratory rate, 18. Examination of the blood showed: hemoglobin, 90 per cent; red cells, 4,960,000; white cells, 16,400; neutrophils, 79 per cent; eosinophils, 2 per cent; lymphocytes, 17 per cent, and endothelial cells,

Fig. 1.—*A* is an anterior view of the patient's neck and the lower part of his face, showing diffuse swelling in the cervical region on the left side. *B* is a lateral view, showing extension of the swelling into the posterior cervical triangle on the left side.

2 per cent. Examination of the urine showed a specific gravity of 1.015, an acid reaction, albumin (2+), a trace of sugar, occasional white blood cells and a small amount of urates.

Roentgenologic Findings.—A roentgenogram of the neck showed swelling and emphysema of the retropharyngeal space anterior to the sixth cervical vertebra. The trachea was displaced forward and to the right. There were marked swelling and diffuse emphysema of the soft tissue of the left side of the neck.

Diagnosis and Course.—The first three days of the patient's illness were marked by an increasing inability to breathe and swallow, and when he was hospitalized the feasibility of surgical intervention, such as evacuation of pus from a possible abscess cavity located either retropharyngeally or laterally, was considered. However, the patient's age, the clinical picture and the roentgenologic findings relegated the possibility of an abscess to the background. Roentgenologically, there was an unquestionable swelling of the soft tissues along the prevertebral muscles and the left cervical region; interspersed were unmistakable air bubbles. The possibility of gas bacillus infection came up, but if such an infection had been present there

would have been, by the time the examination was made, a much greater involvement of the soft tissues around the neck than was actually present. The possibility of a retropharyngeal and cervical emphysema could not be ruled out, but the mechanics of the origin of this condition could not be determined, owing to the patient's inability to open his mouth widely enough to permit a thorough examination. At any rate, masterful inactivity was decided on, and hot compresses were continued for another twenty-four hours.

On January 5 the temperature dropped to normal and the patient could permit his mouth to be opened more widely. The external swelling in the neck extended upward to the mastoid process but otherwise remained unchanged. On the other hand, the throat appeared greatly improved; the uvula was in the midline and the swelling of the left side of the palate had decreased to about half its former size. The left tonsil could be seen. There was a large grayish necrotic, sloughing

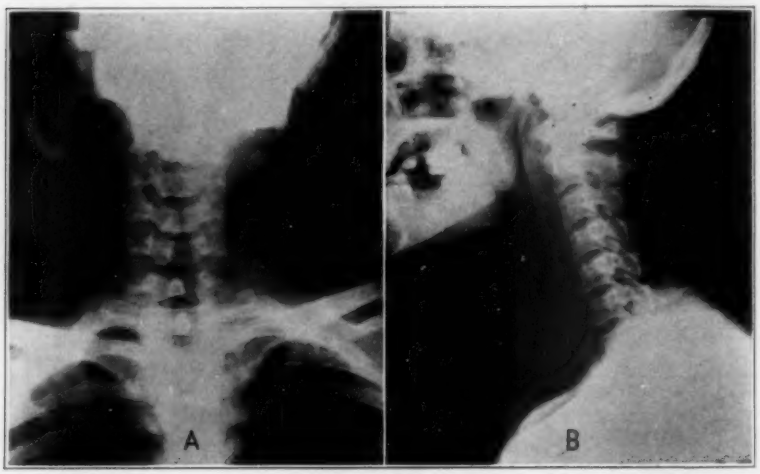

Fig. 2.—*A* shows marked swelling and diffuse emphysema of the soft tissue of the left side of the neck. The trachea is displaced forward and to the right. *B* shows the swelling and emphysema of the retropharyngeal space anterior to the sixth cervical vertebra.

surface on its pharyngeal aspect. It gave off a foul odor. Material for smear and culture was taken from this ulcerated surface.

The smear showed a few pus cells, a few chain streptococci, numerous small gram-negative bacilli, and a few gram-negative filamentous bacilli. Vincent's organisms were not observed. The culture was negative for hemolytic streptococci, staphylococci and Klebs-Loeffler bacilli and positive for nonhemolytic streptococci.

It was apparent that the horn had penetrated the left tonsil and that this tonsillar perforation was the portal of entry for the air which the patient pumped into the cellular tissue in his forceful attempts to breathe. This intake of air was all the more accentuated by his utilizing his accessory muscles of respiration, with which he no doubt was well equipped, considering his asthmatic background.

The throat improved rapidly and was practically normal when the patient left the hospital, eight days after entry. However, a slight cervical swelling on the left side persisted and showed signs of a beginning superficial liquefaction of pus.

ENDAURAL, ANTAURICULAR SURGICAL APPROACH TO THE TEMPORAL BONE

PRINCIPLES INVOLVED IN THIS NEW APPROACH; SUMMARY REPORT OF 1,780 CASES

JULIUS LEMPERT, M.D.
NEW YORK

Surgical operation on the temporal bone through the endaural, antauricular approach consists of three distinct stages. These are:

Stage 1: Operation on the superficial soft parts covering the temporal bone.

Stage 2: Operation on the temporal bone proper.

Stage 3: Surgical treatment and guidance of the membranous and osseous wounds resulting from the first two stages.

The precision with which the technic of these stages is carried out is of the utmost importance to the end result.

STAGE ONE: OPERATION ON THE SUPERFICIAL SOFT PARTS COVERING THE TEMPORAL BONE

Stage 1 consists of the creation of a mobile membranous and extracartilaginous window within the external auditory canal for endaural, antauricular surgical approach to any desired part of the temporal bone.

Since my first report [1] of 165 complete mastoidectomies performed endaurally, it has been my constant aim to improve and perfect the endaural membranous incisions which act as a foundation for the creation of such an endaural membranous window. This study has resulted, by gradual evolution, in the development of the new endaural, antauricular technic.

In creating an endaural, antauricular approach adequate for all types of operations on the temporal bone, the following points must be considered:

1. A series of endaural incisions should be made in the soft parts anterior to the auricle and within the external auditory canal which will lay the foundation for the formation of a membranous endaural

Read at a meeting of the Philadelphia Laryngological Society, Feb. 1, 1938.

1. Lempert, J.: Simple Subcortical Mastoidectomy, Arch. Otolaryng. **7**:201 (March) 1928.

window through which an approach is obtained to the desired surgical field within the temporal bone.

2. The endaural incisions which enter into the formation of the membranous endaural window must always remain extracartilaginous. This is necessary to obviate the possible subsequent development of perichondritis.

3. The membranous endaural window which results from these endaural incisions must afford adequate exposure of the temporal bone and must permit the complete operation to be performed within the desired field with ease and under direct vision.

4. The membranous endaural window not only must be large enough to permit proper exposure for the complete operation, but must be so designed that it involves the least possible amount of sacrifice of the tissues.

5. The membranous endaural window must be made mobile, so that it can, together with the auricle, be freely moved and displaced in any and all desired directions over the temporal bone; this facilitates the operative maneuvers.

6. The membranous endaural window must be so constructed that it will remain wide open throughout the entire postoperative period of healing. This is necessary to insure continuous and free drainage of any infected secretions from the operative wound in the temporal bone.

7. The membranous endaural window must be so made that it will ultimately heal and close without producing any deformity.

To enable the otologist to satisfy all the foregoing requirements a close study of the anatomy of the auricle and the external auditory canal is necessary, and the following anatomic facts are to be noted:

1. Anterior to the superoanterior attachment of the auricle and immediately above and external to the roof of the external auditory canal, there exists a triangular fibrocutaneous space fold or indentation which corresponds to the anatomic landmark where the cartilage which enters into the formation of the auricle is deficient. This triangular fold is bounded posteriorly by the lower end of the helix and anteriorly by the upper part of the tragus. The apex of this triangular space is found at the point at which the helix and the tragus almost meet. Its base is formed by the membranous outermost part of the roof or superior wall of the external auditory canal. This triangular space I have designated the antauricular, suprameatal membranous triangle. When the auricle is held between the index finger and the thumb and pulled upward the antauricular, suprameatal membranous triangle, with all its boundaries is brought prominently into view. The entire triangu-

lar space is filled with connective tissue and contains no important blood vessels or nerves (fig. 1).

After adopting and employing this antauricular, suprameatal membranous triangle as the anatomic basis for the creation of an endaural antauricular approach to all the surgical fields of the temporal bone, I devised three basic types of mobile membranous and extracartilaginous endaural windows. Each of these is most suitable for use in the surgical attack on some particular part of the temporal bone. They are:

1. A window for the endaural, antauricular approach to the mastoid portion of the temporal bone.

2. A window for the endaural, antauricular approach to the mastoid process and the tympanic portion of the temporal bone.

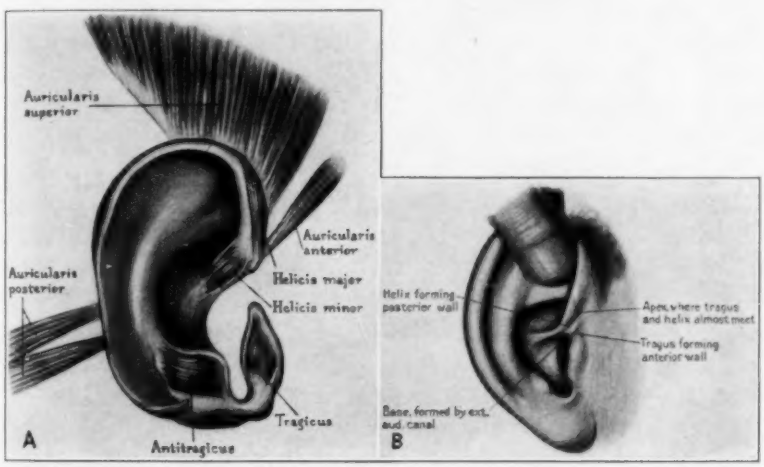

Fig. 1.—*A*, cartilage of the auricle, showing the space between the anterior end of the helix and the tragus where the auricular cartilage is deficient. *B*, antauricular, suprameatal membranous triangle.

3. A window for the endaural, antauricular approach to the mastoid process, the tympanic cavity and the petrous portion of the temporal bone.

TECHNIC OF CREATION OF MOBILE MEMBRANOUS AND EXTRACARTILAGINOUS ENDAURAL WINDOW FOR ENDAURAL, ANTAURICULAR APPROACH TO MASTOID PORTION OF TEMPORAL BONE

In this procedure the membranous portion of the posterior wall of the external auditory canal is sacrificed while the membranous covering of the osseous portion of the posterior wall is preserved.

Control of Bleeding.—Preparatory to the making of the endaural incisions, a 1 : 10,000 solution of epinephrine hydrochloride is injected into the skin and periosteum of the membranous portion of the posterior wall of the external auditory canal internal to the anterior border of the concha. It is also injected into the skin, fibrous tissue and periosteum of the antauricular, suprameatal membranous triangle; this controls bleeding from these tissues, which derive their blood supply from the anterior auricular branch of the superficial temporal artery. The method obviates the devitalization and trauma of tissues which would result from the employment of artery clamps for hemostasis. The negligible bleeding which results from the endaural incisions is further controlled by the subsequent introduction of retractors.

First Endaural Incision.—The membranous part of the external auditory canal is stretched and widened with a narrow-pointed nasal speculum. The first incision is begun in the superoposterior wall at the junction of the membranous and the osseous part of the posterior wall of the external auditory canal. It is then carried downward and outward along the membranous part of the posterior wall of the canal until the lower end of the anterior border of the concha is reached.

Second Endaural Incision.—With the use of the same small nasal speculum, the second incision is begun in the superoposterior wall of the external auditory canal at the point of commencement of the first incision. It is carried outward along the superoposterior wall of the canal up to its outermost limit at the base of the antauricular, suprameatal membranous triangle. The speculum is then discarded, and the auricle is pulled upward with the thumb and the index finger. This brings the antauricular, suprameatal membranous triangle into prominence. The incision is now continued into and through the anterior border of the antauricular, suprameatal membranous triangle, adjacent to the tragus, up to the apex of this triangle, where the helix and the tragus almost meet.

Third Endaural Incision.—The third incision is made by connecting the outer ends of the first two incisions. The anterior border of the concha is brought into prominence by pulling the auricle backward. This third incision is begun at the outer end of the first incision, at the lower end of the anterior border of the concha; it is carried upward along the entire anterior border of the concha to the posterior border of the base of the antauricular, suprameatal membranous triangle. This triangular fibrocutaneous fold is stretched by pulling the auricle upward, and the incision is continued into and through its posterior border immediately adjacent to the helix up to the apex of the antauricular, suprameatal membranous triangle, where the outermost end of the second incision is met (fig. 2).

Comment.—The endaural incisions within the membranous portion of the posterior wall of the external auditory canal are made through the skin and periosteum down to the bony cortex of the mastoid. Those parts of the endaural incisions which extend into the antauricular, suprameatal membranous triangle are made through skin, fibrous tissue and periosteum down to the bony cortex of the posterior root of the zygoma.

These endaural incisions are extracartilaginous. They are so planned and designed in order to obviate the possible occurrence of perichondritis. Nowhere in the path traversed by these incisions does cartilage exist. These incisions are also extramuscular; no muscles are severed by them.

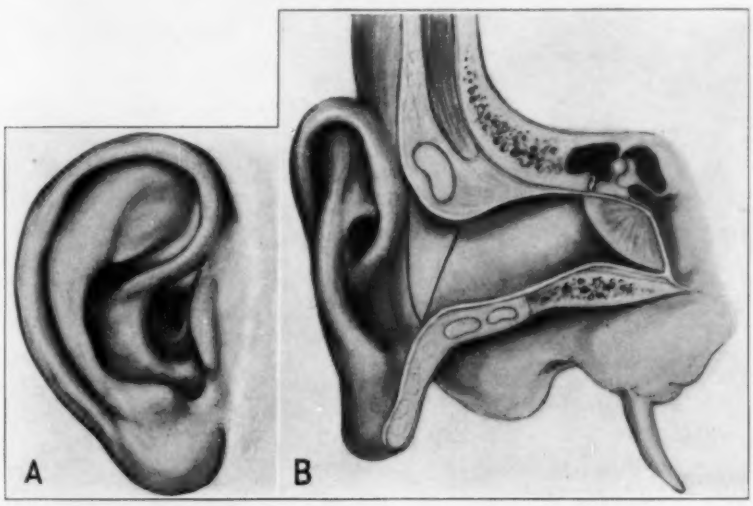

Fig. 2.—Three endaural incisions for the creation of a mobile membranous and extracartilaginous endaural window for endaural, antauricular surgical approach to the mastoid process. *A*, external view; *B*, sectional view.

Resulting from these three endaural incisions there is a triangular flap of membranous tissue.

Removal of the Triangular Membranous Flap.—With the aid of a periosteal elevator, which is inserted into the first endaural incision, the triangular membranous flap is entirely separated and elevated from its attachment to the underlying surface of the bone.

The wedge-shaped, thickest portion of the elevated flap, which occupies the region of the apex of the antauricular, suprameatal membranous triangle, is now grasped with an artery clamp and freed from its underlying attachment with the aid of small curved scissors. The entire membranous triangular flap is removed and discarded. There

is now a membranous extracartilaginous endaural window which exposes to view the outer border of the posterior bony wall of the canal, the anterior border of the outer cortex of the mastoid, the spine of Henle, Macewen's triangle and the outer cortex of the posterior root of the zygoma (fig. 3).

Mobilization of the Membranous and Extracartilaginous Endaural Window.—The postauricular skin and periosteum covering the outer cortex of the mastoid, together with the antauricular skin and periosteum covering the outer cortex of the zygoma, are separated and elevated with a periosteal elevator introduced through the membranous endaural window. The auricle, with all its attachments, is thus freed from the temporal bone. The membranous extracartilaginous endaural window can now be moved and displaced in any direction, exposing to view any desired part of the mastoid process.

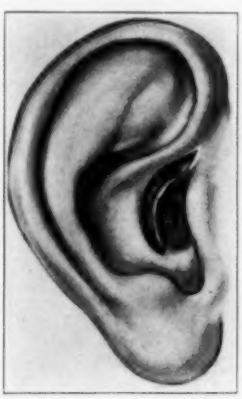

Fig. 3.—Membranous extracartilaginous endaural window for endaural antauricular approach to the mastoid process.

With the introduction of the retractors, which are held in place by an assistant, there is obtained a fully opened, mobile extracartilaginous membranous endaural window which permits complete surgical access to the mastoid portion of the temporal bone through the endaural, antauricular approach.

Comment.—This mobile membranous endaural window permits an adequate exposure of every part of the mastoid portion of the temporal bone not because of the actual surgical field it directly exposes but because of the field it can be made to expose as a result of its mobility. Because the auricle, with its postauricular and meatal attachments, has been freed from the mastoid process and from the zygoma, the mobile membranous extracartilaginous endaural window can be moved with

retractors in situ backward, forward, downward and upward, following the surgeon's technical route in any direction and eventually giving access to the entire mastoid portion of the temporal bone. This mobility of the opened endaural window is not generally comprehended. One trial will convince any surgeon of the easy visualization of the entire operative field obtained by this method.

The otologic surgical procedures which I have performed through the endaural antauricular approach, employing the mobile membranous extracartilaginous endaural window, include:

1. Complete endaural mastoidectomy (simple mastoidectomy).

2. Endaural mastoidoatticotomy (modified radical).

3. Complete endaural exenteration of the contents of the lateral sinus for sinus thrombosis following complete endaural mastoidectomy.

4. Endaural drainage of an abscess in the middle fossa following complete endaural mastoidectomy.

5. Endaural drainage of an abscess in the posterior fossa after complete endaural mastoidectomy.

6. Endaural revision of the mastoid wound left by a Schwartze operation, with plastic repair of the postauricular cavity resulting from the nongranulated mastoid wound.

7. Endaural removal of obstructive exostoses of the bony walls of the external auditory canal.

Advantages Observed.—This mobile membranous and extracartilaginous endaural window for the endaural, antauricular approach to the mastoid portion of the temporal bone is surgically more desirable than the postauricular incision employed with the postauricular approach because it is obtained with much less sacrifice of membranous tissue. Neither the sternocleidomastoid nor the temporal muscle is severed. None of the auricular muscles is injured. No cartilage is sacrificed. Since artery clamps are not employed, the soft tissues are not devitalized.

CREATION OF MOBILE MEMBRANOUS AND EXTRACARTILAGINOUS ENDAURAL WINDOW FOR ENDAURAL, ANTAURICULAR APPROACH TO MASTOID PROCESS AND TYMPANIC PORTION OF TEMPORAL BONE

In this procedure the membranous covering of both the membranous and the osseous portion of the entire posterior wall of the external auditory canal is sacrificed.

Control of Bleeding.—A 1:10,000 solution of epinephrine hydrochloride is injected into the skin and periosteum covering the entire osseous and membranous parts of the posterior wall of the external

auditory canal. It is also injected into the skin, fibrous tissue and periosteum of the antauricular, suprameatal membranous triangle. Bleeding from the anterior auricular branch of the superficial temporal artery, which supplies the anterior portion of the auricle and the membranous part of the external auditory meatus, is thus controlled.

First Endaural Incision.—With the aid of a small narrow-bladed nasal speculum, the membranous portion of the canal is stretched. The first incision is begun in the superoposterior wall of the external auditory canal immediately outside the superoposterior attachment of the tympanic membrane to the tympanic ring. This incision is extended downward and outward, first along the osseous portion and then through the membranous portion of the posterior wall of the canal to the lower end of the anterior border of the concha.

Second Endaural Incision.—The same speculum being used, the second incision is begun from the point of commencement of the first incision, immediately outside the superoposterior attachment of the tympanic membrane to the tympanic ring, and is carried outward along the entire superoposterior wall of the external auditory canal through its entire osseous and membranous portions until the anterior wall of the base of the antauricular, suprameatal membranous triangle is reached. The nasal speculum is discarded, the superoanterior part of the auricle is grasped between the thumb and the index finger of the left hand and the auricle is pulled upward. This stretches and brings into prominence the antauricular, suprameatal membranous triangle. The incision is now extended outward and upward along and through the anterior border of this antauricular, suprameatal triangle, adjacent to the tragus, to the apex of this triangular fold, where the tragus and the helix almost meet.

Third Endaural Incision.—This incision connects the outer ends of the first and second incisions. With the inferoposterior part of the auricle held between the thumb and the index finger of the left hand and the auricle pulled backward, the membranous portion of the posterior wall of the canal is stretched; this brings into prominence the anterior border of the concha, which marks the outermost edge of the posterior wall of the canal. The incision is now begun with the outer end of the first incision at the lower end of the anterior border of the concha, and is carried upward along the entire anterior border of the concha to the posterior wall of the base of the antauricular, suprameatal membranous triangle. The superoanterior part of the auricle is now grasped between the thumb and the index finger of the left hand, the auricle is pulled upward and the membranous antauricular, suprameatal triangle is brought into prominence. The incision is now continued and carried upward along and through the posterior border of the antauricular,

suprameatal membranous triangle, adjacent to the helix, to the apex of this triangle until the outermost end of the second incision is met.

Comment.—These three endaural extracartilaginous and extramuscular incisions form a triangular membranous endaural flap (fig. 4).

Removal of the Membranous Endaural Flap.—The removal of the membranous flap resulting from the endaural incisions just described is accomplished by employing the technic previously outlined. The membranous extracartilaginous endaural window formed by the removal of this flap exposes to view the entire posterior bony wall of the external auditory canal, the anterior border of the outer bony cortex of the

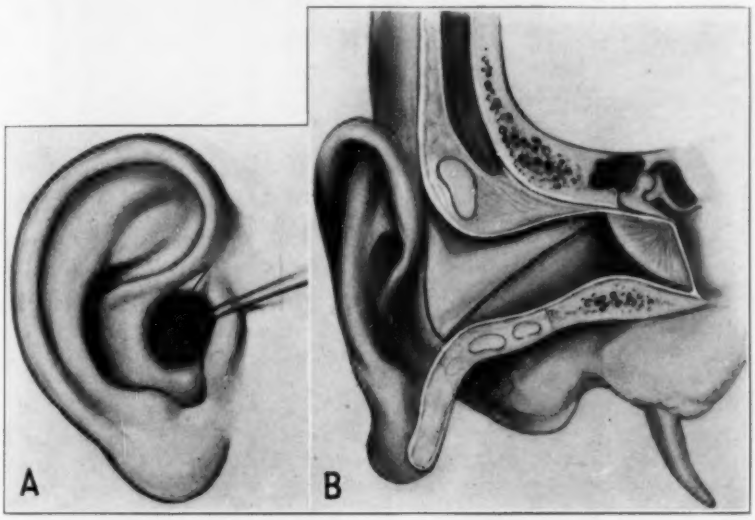

Fig. 4.—Three endaural incisions for the creation of a mobile membranous and extracartilaginous endaural window for endaural, antauricular surgical approach to the mastoid process and the tympanic cavity. *A*, external view; *B*, sectional view.

mastoid, the spine of Henle, Macewen's triangle and the outer bony cortex of the posterior root of the zygoma (fig. 5 *A*).

Mobilization of the Membranous Extracartilaginous Endaural Window.—The postauricular skin and periosteum covering the outer cortex of the mastoid, together with the antauricular skin and periosteum covering the outer cortex of the zygoma, are separated from the bone and elevated with a periosteal elevator introduced through the membranous endaural window. The auricle, with all its attachments, is thus freed from the temporal bone. The membranous extracartilaginous endaural window can now be moved and displaced in any direction to

expose to view any desired part of the mastoid process and the tympanic cavity. After the retractors are inserted there is a fully opened, mobile extracartilaginous membranons endaural window which permits complete surgical access to the mastoid process and the tympanic portion of the temporal bone through the endaural, antauricular approach.

Comment.—The following otologic surgical procedures have been performed by me through the endaural, antauricular approach, the mobile membranous extracartilaginous endaural window being employed:

1. Complete endaural mastoidotympanectomy (radical mastoidectomy).
2. Endaural mastoidotympanolabyrinthectomy.
3. Endaural mastoidotympanoapicotomy.

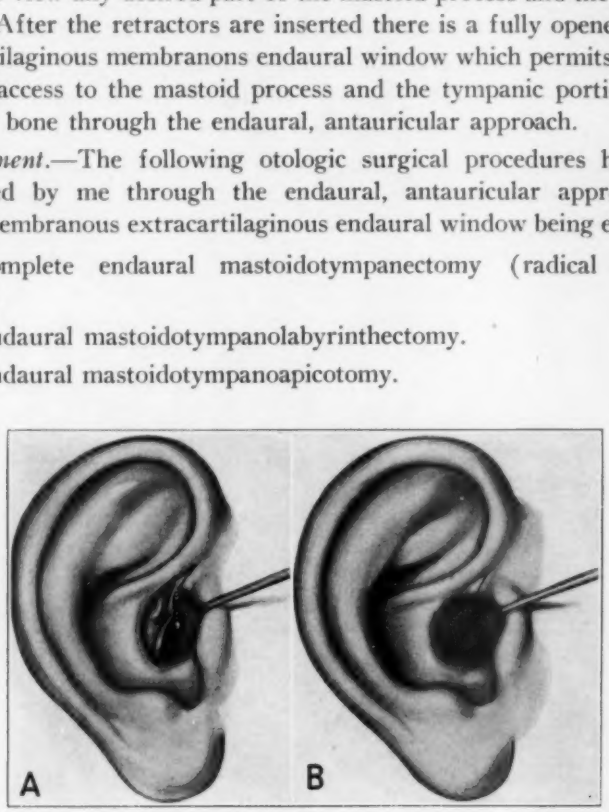

Fig. 5.—*A*, membranous extracartilaginous endaural window for endaural, antauricular surgical approach to the mastoid process and the tympanic cavity. *B*, four endaural incisions for the creation of a mobile membranous and extracartilaginous endaural window for endaural, antauricular surgical approach to the mastoid process, the tympanic cavity and the apical carotid portion of the petrous pyramid.

4. Complete endaural exenteration of the contents of the lateral sinus for sinus thrombosis following complete endaural mastoidotympanectomy.
5. Endaural drainage of an abscess in the middle fossa following complete endaural mastoidotympanectomy.
6. Endaural drainage of an abscess in the posterior fossa following complete endaural mastoidotympanectomy.

Advantages Observed.—In the performance of such operations as have been mentioned, the endaural, antauricular approach through this

mobile membranous endaural window offers to the otologist a fuller and more direct view of this surgical field and more ready accessibility to it. The endaural, antauricular approach facilitates a complete operation within the tympanic cavity more than does the postauricular approach. The endaural incisions employed for the creation of this membranous endaural window are all extracartilaginous. The sternocleidomastoid muscle, the temporal muscle and the auricular muscles are not severed in the creation of the mobile membranous and extracartilaginous endaural window for this approach. This endaural, antauricular approach is surgically more desirable than the postauricular approach because with it only the membranous lining of the posterior wall of the canal is sacrificed; whereas with the postauricular approach, not only are the postauricular skin and periosteum incised, but the entire membranous covering of the posterior wall of the external auditory canal and the concha is sacrificed. Thus the entire membranous structure covering the mastoid portion of the temporal bone is weakened when the postauricular approach to the mastoid and the tympanic portions of the temporal bone is employed.

CREATION OF MOBILE MEMBRANOUS EXTRACARTILAGINOUS ENDAURAL WINDOW FOR ENDAURAL, ANTAURICULAR APPROACH TO MASTOID, TYMPANIC AND PETROUS PORTIONS OF TEMPORAL BONE

In this procedure, in addition to the sacrifice of the membranous lining of the entire posterior wall of the canal, the membranous lining covering the osseous portion of the anterior wall of the external auditory canal is also sacrificed.

Technic.—After the creation of the mobile membranous and extracartilaginous endaural window, as previously described for the endaural, antauricular approach to the mastoid process and the tympanic portion of the temporal bone, an additional incision is made through the membranous lining of the entire anterior wall of the external auditory canal at the junction of its cartilaginous and osseous portions. The skin and periosteum covering the osseous portion of the anterior wall of the external auditory canal are elevated with a subperiosteal elevator and completely removed. This results in exposure of the entire anterior bony wall of the canal, which is formed by the nonarticular part of the mandibular fossa. After this additional exposure there is a mobile membranous and extracartilaginous endaural window which permits complete surgical access, through the endaural, antauricular approach, to the mastoid process, the tympanic cavity and the apical carotid portion of the petrous pyramid (fig. 5 *B*).

Comment.—The following otologic surgical procedures have been performed by me through the endaural, antauricular approach, the mobile membranous and extracartilaginous endaural window being employed:

1. Complete endaural mastoidotympanoapicectomy.[2]

2. Endaural drainage of the cisterna pontis and cisterna interpeduncularis[2] through the posterior wall of the apical carotid portion of the petrous pyramid after complete endaural mastoidotympanoapicectomy.

Advantages Observed.—The endaural, antauricular approach to the apical carotid portion of the petrous pyramid is of great advantage when compared to the postauricular approach because it permits a better and more direct view of the deeply situated surgical field. It also reduces by about 1 inch (2.5 cm.) the depth for the surgical manipulations. This mobile membranous extracartilaginous endaural window is surgically more desirable than the postauricular incision because it sacrifices less tissue. Operation by the endaural, antauricular approach leaves behind a much stronger membranous covering over the temporal bone than does operation by the postauricular approach.

In all surgical operations on the temporal bone which require the removal of a considerable area of the squama, the originally created mobile membranous extracartilaginous endaural window for endaural, antauricular approach may be enlarged by extending the incision from the apex of the antauricular, suprameatal membranous triangle upward along the anterior border of the helix for a distance of ¼ to ½ inch (0.6 to 1.3 cm.) over the squama. This is required in (1) endaural drainage of an abscess in the middle fossa and (2) complete endaural mastoidotympanoapicectomy.

STAGE TWO: OPERATION ON THE TEMPORAL BONE PROPER

TECHNIC

The technic of operation within the confines of the temporal bone should be based on the following surgical principles:

1. At no time should any of the vital anatomic structures within the temporal bone be sacrificed because of incidental injury resulting directly from the use of a particular technic.

2. When an operation is performed for acute or chronic suppuration within any part of the temporal bone, the rule of complete exploration of all the anatomic structures throughout its entire course within the

2. Lempert, J.: Complete Apicectomy (Mastoido-Tympano-Apicectomy), Arch. Otolaryng. **25**:144 (Feb.) 1937.

surgical field must be followed in order to guarantee complete exenteration of the involved bone cells. This is the only sure means of reaching and removing every hidden focus of infection.

3. When an operation is performed on the temporal bone for nonsuppurative functional disorders, the surgical attack must be directly aimed at the area concerned and, as far as possible, should be limited to that area, without unnecessarily exposing any additional structures. Complete exenteration here is not only not essential but actually undesirable.

That complete exenteration is easily accomplished through the endaural, antauricular approach under direct vision has never been disputed by any otologist who has seen this technic used. That such complete exenteration through the endaural, antauricular approach is possible has been verified, and it has also been recorded in the otologic literature by Neumann,[3] of Vienna; Hans Claus,[4] of Berlin; Le Maître,[5] of Paris; Bondy,[6] of Vienna, and Dutheillet de Lamothe,[7] of Paris. Leidler[8] reported, in the *Monatschrift für Ohrenheilkunde,* 9 cases of acute suppurative mastoiditis in which the endaural, antauricular approach was used at the University Hospital of Vienna. In the conclusion of his report he stated: "The Lempert mastoidectomy can be performed in every case of mastoiditis because it is always possible to obtain complete visibility over the entire operative field."

Employing the mobile membranous and extracartilaginous endaural window described, the otologist may easily perform any operation on any part of the temporal bone through the endaural, antauricular approach. He may employ the technical routine to which he is accustomed and the instruments he desires.

In the operations that I have performed within the temporal bone through the endaural, antauricular approach, a technic was employed

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3. Neumann, H., in discussion on Lempert, J.: Einfache subcorticale Mastoidectomie, Monatschr. f. Ohrenh. **63**:1344, 1929.

4. Passow, A., and Claus, H.: Anleitung an den Operationen am Gehörorgan an den Tonsillen und an den Nase, Leipzig, Johann Ambrosius Barth, 1929, pp. 27-28.

5. Le Maître, F., in discussion on Lempert, J.: Mastoidectomie endaurale complete: L'operation de choix dans la mastoidite suppurée aigue, Compt. rend. Soc. franç. d'oto-rhino-laryng., 1931, p. 115.

6. Bondy, G., in discussion on Lempert, J.: Einfache subcorticale Mastoidectomie, Monatschr. f. Ohrenh. **63**:1344, 1929.

7. Dutheillet de Lamothe, G., in discussion on Lempert, J.: Evolution de la mastoidectomie soucorticale simple, Compt. rend. Soc. franç. d'oto-rhino-laryng., 1929, p. 257.

8. Leidler, R.: Bericht über neun der Methode von Lempert ausgeführten Antratomien, Monatschr. f. Ohrenh. **63**:1337, 1929.

which embraced the following principles: (*A*) Immediate and direct opening of the mastoid antrum and exploration of its roof and floor as the first step in any operation on the mastoid portion of the temporal bone. This procedure quickly provides a landmark for orientation which is especially desirable in a surgical field so crowded with vital anatomic structures.

A new technic of opening the mastoid antrum is thus provided which at no time makes any of the anatomic structures liable to accidental injury.

New Technic of Opening the Mastoid Antrum.—An imaginary line is drawn through the superoposterior bony wall of the external auditory

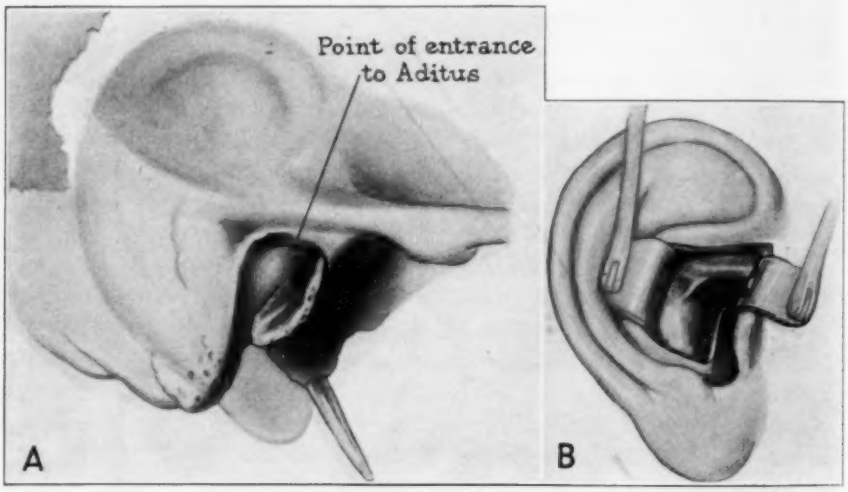

Fig. 6.—*A*, surgical landmark of approach to the aditus and the antrum. *B*, mastoid antrum opened through the new point of entrance.

canal from the spine of Henle to the superoposterior attachment of the tympanic membrane to the tympanic ring. At the junction of the middle and the inner third of this imaginary line, an electrically driven serrated dental bur is applied and directed gently posteriorly and inward for 1 to 2 mm.; the mastoid antrum is reached at once. With the same bur the opening thus created in the superoposterior bony wall of the canal which communicates with the antrum is now enlarged outward toward the spine of Henle and downward along the posterior bony wall of the canal until the antrum is laid wide open and is completely exposed to view (fig. 6).

Comment.—The adoption of this method of entering and exposing the mastoid antrum is the result of considerable study and trial based on a series of observations and anatomic facts.

Macewen,[9] Duplay,[10] Poirer,[11] Politzer,[12] Broca,[13] Whiting[14] and Dench[15] have described various triangles, areas and angles designed to provide an approach through the outer bony cortex of the mastoid into the mastoid antrum with the greatest degree of safety. At the same time, every one of these authors has stated that, though his method is the safest, there are anatomic variations in the position of the lateral sinus and of the middle cranial fossa within the mastoid portion of the temporal bone which make injury to the sinus and the dura almost unavoidable even when the operation is performed by a skilful surgeon according to the advocated method. In 1932 E. B. Burchell,[16] of the New York Eye and Ear Infirmary, in an article entitled "The Temporal Bone and Its Variations" stated: "The last specimen [referring to anatomic specimens which he was describing] demonstrates an extremely far forward and superficial sinus beneath the spine of Henle making it impossible to reach the antrum via the usual surgical route."

Searching for a surgical point of entrance to the mastoid antrum which would be constant and the use of which would never cause accidental injury either to the dura of the middle cranial fossa or to the lateral sinus, no matter how anomalous their anatomic position within the mastoid portion of the temporal bone might be, I observed and recorded the following facts:

1. The mastoid antrum varies in size and may be very small, but it is never absent.

2. No matter how anomalous the position of the lateral sinus within the mastoid process may be, it can never be found herniated into the

9. Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord, Meningitis, Abscess of Brain, Infective Sinus Thrombosis, Glasgow, J. Maclehose & Sons, 1893.

10. Duplay, cited by Lenoir, O.: Contribution to the Study of Antrectomy, Ann. Otol., Rhin. & Laryng. **11**:64, 1902.

11. Poirer, cited by Lenoir, O.: Contribution to the Study of Antrectomy, Ann. Otol., Rhin. & Laryng. **11**:64, 1902.

12. Politzer, A.: Lehrbuch der Ohrenheilkunde für practische Aerzte und Studirende, ed. 3, Stuttgart, Ferdinad Enke, 1893, p. 513.

13. Broca, B. A.: Surgical Anatomy and Operative Surgery of the Middle Ear, translated by Macleod Yearsley, London, Rebman, Ltd., 1901, p. 9.

14. Whiting, F.: Modern Mastoid Operation, Philadelphia, P. Blakiston's Son & Co., 1905, pp. 121 and 126.

15. Dench, E. B.: Complications and Sequellae of Mastoiditis, Laryngoscope **31**:429, 1921; Diseases of the Ear, New York, D. Appleton and Company, 1924, p. 443.

16. Burchell, E. B.: Evaluation of Roentgenology in Otolaryngology: The Temporal Bone and Its Variations, Laryngoscope **42**:921, 1932.

antrum proper. It may be found below the antrum, immediately posterior to or directly external to the antrum or immediately underneath the outer cortex of the mastoid fossa; but it is never found within or anterior to the antrum.

3. No matter how low the middle cranial mastoid fossa may hang, the inner third of the superoposterior bony wall of the canal will always be found to occupy a lower level and to be separated from the middle cranial fossa by the epitympanic space.

4. The distance of the mastoid antrum from the outer cortex of the mastoid varies from 12 to 29 mm.

5. The mastoid antrum lies 1 to 2 mm. posterior and internal to a point in the superoposterior bony wall of the extrenal auditory canal

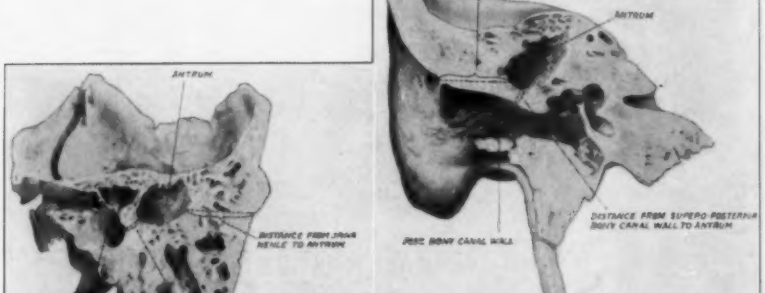

Fig. 7.—Relative distance of approach to the antrum.

represented by the meeting of the middle and the inner third of an imaginary line drawn across this wall (fig. 7).

On the basis of these observations I [1] described a new method of opening the mastoid antrum through a definitely described point in the superoposterior bony wall of the external auditory canal. This method is, I believe, error proof. With this approach to the antrum, neither the middle cranial fossa nor the lateral sinus can ever accidentally be injured, no matter how anomalous an anatomic position these structures occupy within the mastoid portion of the temporal bone. The prominence of the external semicircular canal and the transverse portion of the facial canal are never injured, because they occupy a position anterior and internal to the course of the drill when it enters the antrum.

In proof of my contention that the point of entrance to the antrum which I have chosen is correct and logical I offer the two following phenomena in the pathologic picture of suppurative otitis:

1. Sagging or drooping of the superoposterior wall of the canal. Because the part of the superoposterior bony wall of the canal which separates the mastoid antrum from the external auditory canal is nearer the pus within the antrum than is any other part of the cortex of the mastoid and also because it is thinner than any other part of the cortex, periosteitis of this part of the superoposterior bony wall of the canal is more likely to occur in acute suppurative mastoiditis than is periosteitis over the outer cortex. This produces the sagging or drooping of the superoposterior wall of the external auditory canal.

2. Fistula in the superoposterior bony wall of the canal. It is not unusual in chronic purulent otitis media with cholesteatoma and occasionally in acute suppurative mastoiditis, especially in infants, to discover a fistula in the superoposterior bony wall of the canal which forms a communication between the mastoid antrum and the external auditory canal. The point in the superoposterior bony wall of the canal where the fistula occurs is about the same point which I have chosen for surgical entrance to the antrum as the first step in the technic of operations on the mastoid portion of the temporal bone.

(*B*) Complete exenteration of the mastoid cells. This is strictly done in an operation through the endaural, antauricular approach for suppuration within the temporal bone. As much of the bony outer cortex of the mastoid as is essential to the accomplishment of complete exenteration of the cellular structure of the mastoid process is always removed. In my original publication [1] I advocated exenteration of the mastoid cells with the preservation of most of the outer cortex of the mastoid. After operating thus in the first 165 cases I came to the conclusion that there was no advantage in preserving completely the outer cortex of the mastoid in operations done through the endaural, antauricular approach. In complete endaural mastoidectomy for suppurative mastoiditis, the entire cellular structure of the mastoid is always removed until the inner bony table of the mastoid portion of the temporal bone is completely exposed. The deliberate exposure and tracing of the inner bony table covering the lateral sinus throughout its entire course in the mastoid is employed as a guide and guarantee to complete exenteration of the mastoid cells. All the bony cellular structure lying anterior, posterior, superior and inferior to the lateral sinus is always removed. Special attention is always given to the group of cells lying posterior to the course in the mastoid of the vertical portion of the facial canal and inferoanterior to the lateral sinus. This is known

as the retrofacial cell group. The zygomatic cells are always exenterated until the inner bony table of the posterior root of the zygoma is exposed. Exenteration is never considered complete until the mastoid aspect of the geometric base of the basal labyrinthine portion of the petrous pyramid comes into view. To accomplish this it is often necessary to remove a group of cells covering and surrounding this region; this is known as the basal perilabyrinthine cell area. The importance of exenterating such areas of perilabyrinthine cells when present in a complete mastoidectomy cannot be overemphasized if it is hoped to reduce the frequency of intracranial complications following suppurative mastoiditis.

(*C*) Systematic and deliberate exposure and exploration of all the vital anatomic structures. Throughout their entire course within the mastoid portion of the temporal bone, all the structures are explored religiously in order to prevent accidental injury to them when they are unexpectedly confronted.

Mastoidectomy is considered finished when it presents for inspection:

1. A smooth continuity of the antral tegmen and the mastoid tegmen forming the floor of the middle cranial fossa and the roof of the mastoid portion of the temporal bone.

2. The bony inner table of the posterior root of the zygoma.

3. The mastoid aspect of the geometric base of the basal labyrinthine portion of the petrous pyramid, with the prominence of the horizontal semicircular canal forming the threshold between the mastoid antrum and the aditus.

4. The inner bony table of the mastoid covering the lateral sinus throughout its entire course within the mastoid portion of the temporal bone.

5. The inner bony table of the mastoid covering the dura of the posterior cranial fossa which occupies the region anterior to the entire course in the mastoid of the lateral sinus.

6. The mastoid aspect of the vertical portion of the fallopian canal for the facial nerve.

7. The inner bony table of the mastoid covering the dura of the posterior cranial fossa which occupies the region posterior to the entire course in the mastoid of the lateral sinus.

8. The inner bony table of the mastoid covering the emissary vein.

9. The inner bony table of the mastoid occupying a position inferior to the inferoanterior portion of the course in the mastoid of the lateral sinus which forms the inner bony wall of the mastoid tip (fig. 8*A*).

In a complete endaural mastoidotympanectomy (radical mastoidectomy) for chronic suppuration involving both the mastoid and the tympanic portion of the temporal bone, complete mastoidectomy is first done. The operative wound and the tympanic cavity are then converted into one continuous mastoidotympanic cavity. The infected contents from within the tympanic cavity are removed. In the exenteration of the tympanic cavity the vital anatomic structures within it are deliberately exposed completely in order to avoid accidental injury to

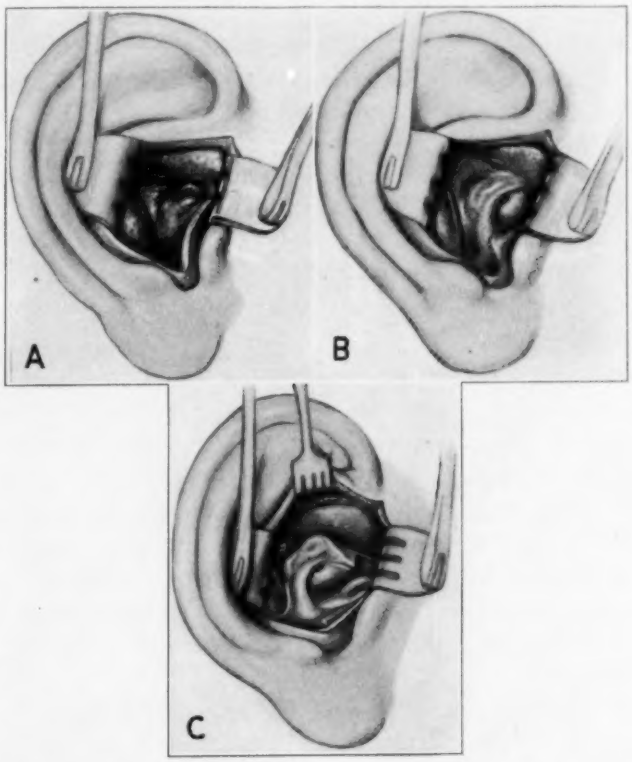

Fig. 8.—*A*, complete endaural mastoidectomy (simple mastoidectomy); *B*, complete endaural mastoidotympanectomy (radical mastoidectomy); *C*, complete endaural mastoidotympanoapicectomy.

them. After the removal of the cholesteatomatous mass, which usually includes the remnants of the incus and the malleus from the tympanic cavity, the first anatomic structure deliberately exposed throughout its tympanic course is always the transverse tympanic portion of the bony canal for the facial nerve, which occupies a position anterosuperior to the prominence of the horizontal semicircular canal and posterosuperior

to the oval window. The transverse portion of the facial canal when thus exposed and under direct vision is safe from accidental injury. After all the infected contents have been removed from the epitympanic space, the tympanic cavity and the hypotympanic space, the tubal cells from the region of the tympanic orifice of the eustachian tube are always exenterated. The oval window, with the stapes in situ, is brought into view by removing the posterior margin of the tympanic ring. With the transverse portion of the facial canal, the oval window, the promontory of the cochlea and the tympanic orifice of the eustachian tube completely exposed to view, the cochleariform process is destroyed, and the tensor tympani muscle is removed from its bony semicanal.

Thus the transverse portion of the bony canal for the facial nerve, the oval window and the promontory of the cochlea are deliberately and early exposed to view in order to avoid injury to them, because they are the vital anatomic structures in this region of the temporal bone and injury to them may prove disastrous.

In a completed mastoidotympanectomy there is always demonstrable:

1. The entire inner bony table forming the floor of the middle cranial fossa, consisting of the mastoid tegmen, the antral tegmen and the tympanic tegmen.

2. The entire inner bony table covering the lateral sinus throughout its course within the mastoid portion of the temporal bone.

3. The inner bony table forming the anterior perisinal aspect of the posterior cranial fossa (Trautmann's triangle).

4. The inner bony table forming the posterior perisinal aspect of the posterior cranial fossa.

5. The floor of the antrum and aditus.

6. The mastoid aspect of the geometric base of the basal labyrinthine portion of the petrous pyramid, with the eminence of the external semicircular canal.

7. The remaining lower limit of the tympanic portion of the posterior bony wall of the external auditory canal, enclosing the descending vertical portion of the facial nerve within the fallopian canal (commonly known as the facial ridge).

8. The transverse tympanic portion of the facial nerve in its bony canal.

9. The oval window, with the stapes in situ.

10. The promontory of the cochlea.

11. The semicanal for the tensor tympani muscle.

12. The tympanic orifice of the eustachian tube.

13. The floor of the hypotympanic space leveled down to and continuous with the bony floor of the external auditory canal (fig. 8*B*).

The same surgical principles are applied whenever complete endaural mastoidotympanoapicectomy is performed for suppuration of the temporal bone extending into the apical carotid portion of the petrous pyramid. The internal carotid artery in its carotid canal is employed as a guide to the complete exenteration of all the pathologic processes within the apical carotid portion of the petrous pyramid. With this technic of exposing and following the internal carotid artery throughout its entire course within the apical carotid portion of the petrous pyramid, all the cellular structure from within the apical carotid portion of the petrous pyramid is removed until its entire inner bony table which forms the wall of the posterior cranial fossa anterior to the internal auditory meatus and the inner bony table forming its floor are exposed. The entire dura of the middle cranial fossa of the temporal lobe, which rests on the anterosuperior surface of the apical carotid portion of the petrous pyramid, is always completely exposed. This area is explored up to the geometric apex of the apical carotid portion of the petrous pyramid, where the internal carotid artery ends its transverse course and bends upward to make its exit from the petrous pyramid. The employment of the internal carotid artery as a guide to complete exenteration makes accidental injury to this important vessel absolutely avoidable (fig. 8 *C*).

The rule of deliberate, systematic and complete exposure of all the vital anatomic structures encroaching on the surgical field has been applied and strictly adhered to in all surgical procedures attempted on the temporal bone. Such a technic prevents accidental injury to these anatomic structures. A focus of infection, no matter how remotely situated, cannot possibly escape detection and removal when such a technic is employed.

STAGE THREE: SURGICAL MANAGEMENT OF MEMBRANOUS AND OSSEOUS WOUNDS RESULTING FROM FIRST TWO STAGES OF ENDAURAL, ANTAURICULAR OPERATION ON TEMPORAL BONE

The surgical management of the membranous and osseous wounds resulting from operations on the temporal bone through the endaural, antauricular approach is based on surgical principles which guarantees:

1. Constant unimpeded drainage of the osseous wound throughout the period of healing.

2. Constant visibility of the osseous wound to permit inspection and guidance of the process of repair in order to obtain the desired healing.

3. A period of healing and convalescence resulting in the least possible social and economic inconvenience to the patient.

4. An ultimately healed osseous and membranous wound whose appearance approaches cosmetically as nearly as possible its preoperative appearance.

SURGICAL MANAGEMENT OF POSTOPERATIVE OSSEOUS AND MEMBRANOUS WOUNDS RESULTING FROM OPERATION ON MASTOID PORTION OF TEMPORAL BONE THROUGH ENDAURAL, ANTAURICULAR APPROACH

In complete endaural mastoidectomy (simple mastoidectomy) the membranous lining covering the osseous portion of the posterior wall of the canal is left intact. The mobile membranous extracartilaginous endaural window employed for the performance of complete endaural mastoidectomy is never sutured and remains completely open. The osseous mastoid wound is neither packed nor drained through the insertion of any foreign substance. Drainage naturally continues unimpeded through the open membranous endaural window. As soon as healthy granulations fill the entire cavity of the osseous wound, the membranous endaural wound within the membranous portion of the posterior wall of the external auditory canal heals and closes. The completely healed posterior membranous wall of the external auditory canal closely approaches cosmetically its preoperative state. It is difficult to distinguish between an ear which has been operated on and an ear which has not. The period of healing varies from two to six weeks. Superficial dressings and bandages are worn only for the first four days after the operation. For the rest of the period of healing sterile cotton within the canal is the only external dressing needed. The occasional fistulization of the posterior wall of the canal, occurring as a result of an arrested process of healing within the bony mastoid cavity (which was encountered when the membranous covering of the entire posterior wall of the canal was sacrificed in the formation of the mobile membranous extracartilaginous endaural window for the endaural mastoidectomy), is done away with by adoption of the new technic whereby the membrane covering the osseous portion of the posterior wall of the canal is left intact.

Postoperative infections of the soft tissues covering and surrounding the temporal bone, such as eczema, cellulitis, cervical adenitis and infections of the sternocleidomastoid and temporal muscles, do not occur after complete endaural mastoidectomy.

Historical Review.—The tremendous advantages enjoyed by the patient as a result of this treatment can be obtained only when the endaural antauricular approach is employed in operations on the temporal bone. This fact is best shown by a brief review of the history of the general treatment of the postoperative postauricular mastoid wound.

From 1879, when Schwartze first described his technic for mastoidectomy, until 1910 it was the general practice of otologists to leave the postauricular mastoid wound wide open and to pack it with gauze (preferably iodoform gauze), allowing the wound to heal by granulation. The reason for this was the well recognized necessity for maintaining constant drainage of the antrum and the mastoid wound in order to prevent retention of pus and consequent intracranial complications. The results obtained with this after-treatment of the Schwartze mastoid wound were disappointing when compared with those obtained after surgical procedures elsewhere in the body. Complications accompanying the healing process, such as eczema, erysipelas, cellulitis, cervical adenitis and infections of the sternocleidomastoid muscle and the temporal muscle, were not infrequent. The prolonged period of professional and social incapacity of the patient resulting from the Schwartze postoperative treatment of the mastoid wound, varying from three months to two years, did not harmonize with the modern advance in general surgery and therefore became a source of no little concern to otologists. The final result, the completely healed postauricular wound presenting an ugly depressed scar, collapse of the auditory canal, a disfiguring dermatized cavity of varying size or an arrested nondermatized nongranulating postauricular cavity of varying size, was unsatisfactory. As a result of this general dissatisfaction, Reik,[17] in 1910, suggested the blood clot method of healing the postauricular mastoid wound. This method of closure of the mastoid wound, after having been tried by many otologists, fell into disuse because of intracranial complications resulting from the frequent infection of the blood clot without a chance for its drainage. Otologists, however, have since adopted various modifications of the Reik treatment of wounds, such as:

1. Drainage for twenty-four hours followed by secondary suture.

2. Suture of the skin and periosteum, with the insertion of a drain at the lower end of the wound (Kerrison[18]).

3. Partial suture of the skin and periosteum and drainage of the lower third of the mastoid wound.

4. Closure of the mastoid wound above and below, with the central part left open and drained with a glass tube (Uffenorde[19]).

17. Reik, H. O.: Primary Union After Mastoidectomy, Bull. Johns Hopkins Hosp. **21**:5, 1910.

18. Kerrison, P. D.: Mastoid Operation of Ten Years Ago and Today, J. A. M. A. **68**:273 (Jan. 27) 1917.

19. Uffenorde, in Passow and Claus,[4] p. 26.

5. Complete closure of the postauricular wound and counterdrainage through the posterior meatal wall by way of counterincision in the meatus.

Of all these modifications of the blood clot method of treatment of the postauricular mastoid wound, not one can lay claim to being applicable in every case, and not one of these methods when employed even in a suitable case is entirely free from risk. Although any one of them may result in a more quickly and better healed wound than the wide open method of treatment, these results are not worthy of consideration if they have to be obtained at the expense of even an occasional intracranial complication arising directly from any method in which the chief surgical principle of drainage is either wholly or partially sacrificed. The mastoid wound if exenteration is complete is a smooth cavity with elevations and depressions caused by the anatomic formation of the inner table covering the lateral sinus and dividing the mastoid cavity into several compartments. With postauricular suture of the periosteum and skin, these compartments become isolated, and intercommunication between them is impossible. While one compartment may be drained, the other remains shut off from drainage. Some otologists who practice partial or complete closure of the mastoid wound argue that it is worth trying and that if the patient shows signs of retention of pus or of infection manifested by a rise in temperature, the wound can be readily opened, and wide open drainage can be established; thus, they say, there is nothing to lose and all to gain. This argument is entirely fallacious. The risk is not to be minimized. Often the first rise in temperature is the beginning of an intracranial complication which cannot be checked by establishing wide open drainage. An occasional neat scar or possible rapid healing obtained at such a price is not worthy of consideration.

In a discussion of the after-treatment and results of mastoid operations at the annual meeting of the British Medical Association in 1926, Jenkins,[20] of London, in his address entitled "The Schwartze Operation for Acute Mastoiditis: After-Management of the Wound," stated: "The walls of the mastoid wound, no matter how carefully cleaned or what antiseptics are used, must be regarded as infected with an organism which has been responsible for the acute mastoiditis and is capable of causing still further extension of the disease in spite of the resistance of the individual." He emphasized the importance of wide open drainage of the mastoid wound. He condemned primary or partial suture of the wound without proper drainage by stating that even in his selected cases primary or partial suture without proper drainage has occasionally

20. Jenkins, C. J.: The Schwartze Operation for Acute Mastoiditis: After-Management of the Wound, Brit. M. J. **2**:1153, 1926.

failed. He said he felt that the risk and disturbance to the patient were too great and that the results, even when successful, were not always satisfactory. He advocated leaving the whole postauricular incision wide open and packing all parts of the wound lightly with iodoform gauze. Admitting that the employment of this method may seem a prolonged after-treatment, Jenkins said he strongly adheres to it, with the full knowledge that some of the wounds he treats by this method would have healed with the primary suture. He pointed out that it is the occasional case in which serious consequences result from reinfection resulting from lack of drainage that impels him to continue using the wide open method, which is always safe.

Neumann,[21] of Vienna, in his address entitled "Results of Operations for Mastoiditis," read before the British Medical Association in 1926, stated that after thorough exenteration of all the mastoid cells in every case of acute suppurative mastoiditis as taught and practiced in his clinic, the operative cavity left is naturally a large one and that therefore the after-treatment is somewhat prolonged because the wound is left wide open. The resulting scar is usually large, but this is counterbalanced by the good results obtained from the complete exenteration and the wide open drainage. Neumann argued that if sacrifice must be made in the after-treatment of the mastoid wound, he would rather have an unpleasant-looking scar than subject the patient to the risk of postoperative complications resulting from reinfection by sacrificing thoroughness of exenteration or violating the surgical principle of unimpeded drainage. This position was justly taken. Neumann closed his address with the following remark: "As in other questions of medicine, we do not claim that the last word has been spoken, but we propose to persist in this method and practice until possibly other methods and further research compel us to adopt other treatment."

Being perfectly in accord with these observations of Jenkins and of Neumann, because of my own experience with 1,500 Schwartze postauricular mastoidectomies, I concluded that as long as unimpeded continuous drainage of the mastoid cavity remains indisputably essential to the life of the patient, neither complete nor partial closure of the postauricular mastoid wound is to be considered. With admission of the fact that the postauricular wound is best left wide open after complete mastoidectomy, the desire to attempt to overcome the necessary hideous postauricular scar or depression, as well as the shocking preponderance of various traumatic complications resulting from the Schwartze open wound treatment, became absolutely hopeless. It was my dissatisfaction

21. Neumann, H.: Results of Operations for Mastoiditis, Brit. M. J. 2:1157, 1926.

with the postoperative treatment of the postauricular mastoid wound and the results obtained therefrom that prompted me to seek a different surgical approach to the temporal bone. I sought an avenue of approach which would entail less trauma to the soft tissues and less sacrifice of them, one which would result in a wound that could be left open to maintain the essential continuous drainage without danger of complications in the soft parts. I sought a method by which there would be left a wound which would ultimately heal and close without leaving hideous deformities and which when completely healed would present an appearance cosmetically approaching the preoperative appearance. To meet these requirements the endaural incisions have been designed and created. They form a series of mobile membranous and extracartilaginous endaural windows for the endaural, antauricular approach in connection with all operations on the temporal bone.

Comment.—This postoperative treatment of the osseous and membranous wounds after complete endaural mastoidectomy offers the following advantages:

1. The membranous endaural wound cannot be closed and will not close of itself until the mastoid cavity is fully granulated. This permits constant inspection of the mastoid cavity during the entire period of healing.

2. The membranous endaural wound, because of its inability to close, maintains constant unimpeded drainage of the infected osseous mastoid wound.

3. Absence of postoperative complications arising in the membranous wound results because of the fact that with the endaural, antauricular approach there is a comparatively small sacrifice of soft parts. No muscles are severed.

4. The membranous endaural wound closes by itself as soon as the osseous mastoid wound is healed.

5. When the membranous endaural wound completely heals, there is close resemblance cosmetically to the preoperative appearance. This cosmetic result is always obtainable without sacrificing either wholly or partially the all-important surgical principle of drainage.

6. The completely healed wound left after complete endaural mastoidectomy offers greater resistance to future infection of the middle ear [22] because it is anatomically stronger than the healed wounds left after other procedures.

7. The period of healing following complete endaural mastoidectomy is much shortened. It results in no social or economic inconvenience

22. Lempert, J.: Die Verhütung von rezidivierender Mastoiditis und die endaurale Operationsmethode bei dieser, Monatschr. f. Ohrenh. **63**:185, 1929.

to the patient because the superficial dressing is worn for only four days after the operation.

The substitution of the endaural, antauricular surgical approach to the mastoid process for the postauricular approach provides a logical and accurate surgical procedure, with the hitherto vexatious problem of after-care solved by very reason of the logical nature and correctness of the initial technic employed.

SURGICAL MANAGEMENT OF POSTOPERATIVE OSSEOUS AND MEMBRANOUS WOUNDS RESULTING FROM OPERATION ON MASTOID AND TYMPANIC PORTIONS OF TEMPORAL BONE THROUGH ENDAURAL, ANTAURICULAR APPROACH

The membranous endaural wound resulting from complete endaural mastoidotympanectomy (radical mastoidectomy) is left completely open. Neither the osseous mastoid wound nor the tympanic cavity is packed. Unimpeded drainage is thus constantly maintained. A superficial dressing and bandage is applied daily for the first four days after the operation and is discarded on the fifth day. The process of granulation is allowed to continue until the inner bony wall of the mastoidotympanic cavity is completely covered by a thin layer of granulations. By continuous cauterization the granulations are prevented from proliferating beyond the desired thickness necessary to cover the bony surface. Through careful guidance the process of granulation is halted, and epidermization is encouraged to extend and spread from the edges of the epidermis lining the remaining lower portion of the anterior wall and the floor of the external auditory canal until the entire inner wall of the mastoidotympanic cavity is covered with a thin layer of epidermis.

Advantages of This Treatment of the Wound.—If the employment of the thick plastic flap is abandoned and if nature is allowed to do the epidermization a better auditory result is obtained. No plastic procedure being employed, there is no sacrifice of concha, and a better cosmetic result follows. The healed membranous wound is stronger because when the endaural, antauricular approach is employed, only the membranous lining of the posterior wall of the canal is sacrificed, whereas with the postauricular approach the postauricular skin and periosteum, the membranous lining of the entire posterior wall of the external auditory canal and part of the concha are sacrificed. Cosmetically, the appearance of the parts after complete endaural, antauricular mastoidotympanectomy approaches their appearance before the operation.

SURGICAL MANAGEMENT OF POSTOPERATIVE OSSEOUS AND MEMBRANOUS WOUNDS RESULTING FROM OPERATION ON MASTOID, TYMPANIC AND PETROUS PORTIONS OF TEMPORAL BONE THROUGH ENDAURAL, ANTAURICULAR APPROACH

The membranous endaural wound resulting from complete endaural mastoidotympanoapicectomy is left wide open in order to guarantee unimpeded drainage from the apical carotid portion of the petrous pyramid. The wound in the temporal bone is not packed. Granulations are allowed to form and fill the osseous wound within the apical carotid portion of the petrous pyramid up to the anterior end of the cochlear promontory. The remaining mastoidotympanic wound is treated in the same manner as after mastoidotympanectomy. The inner bony wall of the tympanic cavity is allowed to granulate sufficiently to form a thin covering, and then epidermization is encouraged. The resulting healed wound resembles that following complete mastoidotympanectomy.

Advantages of This Treatment of the Wound.—Since no plastic procedure is employed and since the tympanic cavity is allowed to heal by epidermization, a better auditory result is obtained. Owing to the comparatively small sacrifice of soft tissues, a stronger healed membranous wound results from the endaural mastoidotympanoapicectomy. The appearance of the ultimately completely healed wound approaches cosmetically the appearance before the operation.

RÉSUMÉ OF THE DATA IN 1,780 CASES

Surgical Conditions.—The 1,780 operations most recently done by me for suppurative disease involving all parts of the temporal bone and for every possible intracranial complication arising therefrom have been performed by the endaural approach. The original technic described in the report of the first 165 cases [1] was improved on, and the new endaural, antauricular surgical approach to the temporal bone was adopted and employed exclusively in the 1,615 cases. The data may be summarized as follows:

1. Complete endaural mastoidectomy for acute suppurative mastoiditis was performed in 1,595 cases.

2. Endaural antroatticotomy (modified radical) was performed for chronic purulent otitis media without cholesteatoma in 10 cases.

3. Complete endaural mastoidotympanectomy for chronic purulent otitis media with cholesteatoma was performed in 129 cases.

4. Endaural mastoidotympanolabyrinthectomy for acute progressive suppurative labyrinthitis with invasion of the cerebrospinal fluid was performed in 5 cases.

5. Endaural mastoidotympanoapicotomy was performed for drainage of suppurative apicitis in 1 case.

6. Complete endaural mastoidotympanoapicectomy was performed for complete exenteration of the apical carotid portion of the petrous pyramid in suppurative apicitis in 7 cases. In 5 cases acute suppurative meningitis accompanied the suppurative apicitis previous to operation.

7. Complete endaural exenteration of the infected contents of the lateral sinus in sinus thrombosis was accomplished in 23 cases.

8. Endaural drainage of an abscess of the temporosphenoidal lobe by way of the middle fossa was established in 3 cases.

9. Endaural drainage of a cerebellar abscess by way of the posterior fossa was established in 2 cases.

10. Complete endaural resection of the entire temporal bone for sarcoma was performed in 1 case.

11. Complete endaural mastoidotympanoapicectomy was performed to establish supratentorial drainage of the infected contents of the cisterna interpeduncularis, together with infratentorial drainage of the infected contents of the cisterna pontis through the wall of the posterior fossa of the apical carotid portion of the petrous pyramid for acute bacterial suppurative meningitis, in 4 cases.

Roentgenography.—A roentgenologic study of the temporal bones was made in each of the 1,780 cases before surgical intervention was undertaken.

Age Incidence.—The ages of the patients operated on were as follows:

Age, Years	No. of Cases
Below 1	97
1– 5	621
5–10	464
10–20	223
20–30	133
30–40	94
40–50	75
50–60	47
60–70	20
70–80	6
Total	1,780

Causative Organisms.—Cultures made from the pus found within the temporal bone at the time of surgical intervention in the 1,780 cases revealed:

	No. of Cases
Streptococcus haemolyticus	1,226
Streptococcus anhaemolyticus	280
Streptococcus mucosus (Pneumococcus type III)	79
Staphylococcus aureus	83
Streptococcus viridans	2
Pneumococcus type IV	10
Diphtheroid bacilli	39
Mixed infection	31
No growth	20
Bacillus pyocyaneus	10
Total	1,780

Cultures made of the blood taken from patients suffering from sinus thrombosis revealed:

	No. of Cases
Streptococcus haemolyticus	18
Streptococcus mucosus (Pneumococcus type III)	3
No growth	2

Cultures made of the cerebrospinal fluid taken from patients suffering from acute suppurative meningitis revealed:

	No. of Cases
Streptococcus haemolyticus	22
Streptococcus mucosus (Pneumococcus type III)	4
No growth	2

Pathologic Process.—The following pathologic processes involving the bony structure of the mastoid portion of the temporal bone were encountered:

Pathologic Processes		No. of Cases
Necrosis of bone	Complete cell necrosis	467
	Partial cell necrosis	134
	Basal perilabyrinthine cell necrosis in region of mastoid aspect of geometric base of petrous pyramid	117
	Necrosis of vertical portion of facial canal	17
	Retrofacial cell necrosis	165
	Complete necrosis of outer cortex	12
Formation of abscess of bone	Complete abscess cavity	623
	Abscess of tip	390
	Abscess in Trautmann's triangle	261
	Abscess in region of emissary vein	38
	Abscess in posterior root of zygoma	127

Perforation of bone	Perforation of outer cortex in region of Macewen's area, with subperiosteal abscess	189
	Perforation of outer cortex in region of emissary vein, with subperiosteal abscess	21
	Perforation of center of outer cortex, with subperiosteal abscess	12
	Perforation of outer cortex in region of tip, with subperiosteal abscess	14
	Perforation of outer cortex of zygoma, with subperiosteal zygomatic abscess	37
	Perforation of posterior bony wall of canal	21
	Perforation in median wall of mastoid tip with Bezold abscess	14
Pathologic processes involving course of lateral sinus in mastoid	Perisinal abscess	227
	Obliterating thrombus within lumen of lateral sinus	13
	Abscess formation within lumen of sinus	5
	Partial thrombosis within lumen of sinus wall	3
	Perforated sinus wall with broken-down infected clot within lumen of sinus	2
Pathologic processes involving middle cranial fossa mastoideae	Epidural abscess	76
	Subdural abscess	3
	Abscess of temporal lobe	2
Pathologic processes involving posterior cranial fossa mastoideae	Epidural abscess	11
	Cerebellar abscess	2

The following pathologic processes were encountered within the tympanic portion of the temporal bone:

Pathologic Processes		No. of Cases
Within tympanic cavity	Cholesteatomatous mass	104
	Polypi and granulations	34
	Necrosis of cells around eustachian tube	28
Involving middle cranial fossa tympani	Epidural abscess	10
	Abscess of temporal lobe	1
Involving inner wall of tympanic cavity formed by tympanic wall of basal labyrinthine portion of petrous pyramid	Necrosis of transverse tympanic portion of bony canal for facial nerve, with facial nerve lying exposed	8
	Necrosis of semicanal for tensor tympani muscle	9
	Fistula of cochlear promontory	1
	Infracochlear perilabyrinthine fistula	2
	Supracochlear perilabyrinthine fistula	2

The following pathologic processes involved the basal labyrinthine portion of the petrous pyramid:

Pathologic Processes	No. of Cases
Perilabyrinthine cell fistula on superior bony middle fossa surface anterior to arcuate eminence of superior semicircular canal, with epidural abscess	3
Perilabyrinthine cell fistula on bony posterior fossa surface anterior to the posterior semicircular canal, with epidural abscess	4
Fistula of external semicircular canal	27
Free pus in membranous labyrinth found during labyrinthectomy	2
Liquefaction necrosis of jugular dome	2

The following pathologic processes involved the apical carotid portion of the petrous pyramid:

Pathologic Processes	No. of Cases
Necrosis of apical cells within apical carotid portion	7
Abscess cavity or empyema of apical carotid portion	5
Pericarotid abscess	2
Absence of outer bony wall of carotid canal as result of necrosis	4
Cortical perforation in bony anterosuperior middle fossa surface with epidural abscess	2
Cortical perforation in bony posterior fossa surface anterior to internal auditory meatus, with epidural abscess	3
Cortical perforation in bony inferior surface (base of skull) of apical carotid portion of petrous pyramid	1
Fistula between superoanterior wall of cochlear promontory and bend of vertical tympanic portion of internal carotid canal	1

Neoplasm of the temporal bone was observed in 1 case; lymphosarcoma involved the entire temporal bone.

Mortality.—In 37 of the 1,780 cases death occurred, giving a mortality rate of 2.08 per cent. The causes of death were as follows:

Cause of Death	No. of Cases
Acute suppurative meningitis, present and recognized at time of entry to hospital and previous to surgical intervention	17
Acute suppurative meningitis becoming manifest subsequent to operation on the temporal bone	6
Rupture of temporosphenoidal abscess into ventricles (confirmed by autopsy)	1
Perforated gastric ulcer with gastromalacia as a terminal cause in temporosphenoidal abscess; successfully explored and drained (confirmed by autopsy)	1
Septic pneumonia resulting from septic lateral sinus thrombosis	3
Uremia	1
Cavernous sinus thrombosis complicating lateral sinus thrombosis	1
Severe general sepsis following sinus thrombosis	3
Cerebellar abscess (confirmed by autopsy)	1
Abscess of pons (confirmed by autopsy)	1
Ether pneumonia	1
Pulmonary edema	1
Total	37

SUMMARY

Endaural, antauricular operation on the temporal bone is not performed through the external auditory canal but through a widely opened mobile membranous and extracartilaginous endaural window which, together with the auricle, may be moved and displaced in every desirable direction over the temporal bone.

A new anatomic landmark is described and designated as the antauricular, suprameatal membranous triangle. The surgical employment of this triangular membranous space makes possible the endaural, antauricular approach in all surgical attacks on the temporal bone.

Many technical difficulties encountered with the postauricular surgical approach to the temporal bone, some of which are often insurmountable to the less experienced and conquerable only by the extremely skilful surgeon, are naturally solved by the very technic of the newly described endaural, antauricular surgical approach.

A careful comparative study of the postoperative course, the convalescence, the period of healing and the ultimate end results obtained in the 1,780 consecutive cases in which operation was performed by me through the endaural, antauricular surgical approach, together with the careful observation of the many advantages enjoyed by my patients as a result of the adoption of this new technic, more than justifies my abandonment of the postauricular approach to the temporal bone.

AIR CELLS OF PETROUS PORTION OF TEMPORAL BONE IN A CHILD FOUR AND A HALF YEARS OLD

A STUDY BASED ON WAX PLATE RECONSTRUCTIONS

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The structure of the mastoid portion of the temporal bone has been thoroughly studied, and the nature and extent of its pneumatization are now well understood. The form, arrangement and relationships of the cells in the petrous portion have not been described with equal thoroughness. The present study finds its justification in the current demand for reliable information concerning the anatomy of this important subdivision of the temporal bone.

Siebenmann [1] made the first painstaking study of the air cells of the temporal bone; by means of corrosion preparations he demonstrated the shape and connections of the mastoid cells and of certain lesser pneumatic spaces situated beyond the mastoid region, stating that they penetrate everywhere with the exception of the squama and the anterior wall of the external auditory meatus. Illustrations of the corrosion specimens (see also Bezold and Siebenmann [2]) are still being copied in textbooks of anatomy and otology.

This paper was read first at a meeting of the American Laryngological, Rhinological and Otological Society, Chicago, Jan. 11, 1937.

From the Departments of Anatomy and Otolaryngology of Northwestern University Medical School; contribution no. 255 from the former; an investigation conducted under the auspices of the Central Bureau of Research of the American Otological Society.

1. Siebenmann, F.: Die Korrosions-Anatomie des knöchernen Labyrinthes des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1890; Mittelohr und Labyrinth, in von Bardeleben, K.: Handbuch der Anatomie des Menschen, Jena, Gustav Fischer, 1897, vol. 5, pp. 195-324.

2. Bezold, F., and Siebenmann, F.: Textbook of Otology, Chicago, E. H. Colegrove Co., 1908.

It is now an established fact that the degree of pneumatization of the petrous pyramid varies greatly in different subjects. Belinoff and Balan,[3] after a study of gross sections, reported that 78 per cent were pneumatic; Kraus,[4] utilizing roentgenograms, found that approximately 24 per cent of the pyramids studied were either thoroughly or moderately pneumatized; Glick's [5] figure was 43 per cent. Still other percentages were reported by Myerson, Gilbert and Rubin [6] and by Friesner, Druss, Rosenwasser and Rosen.[7] Jones,[8] observing that the various percentages based on examination of roentgenograms have been proved by microscopic examination to be inaccurate, concluded that only after hundreds of temporal bones have been carefully examined by the latter method can tables of percentages be accurately compiled. It may be added that any method allowing only gross examination is entirely inadequate; the intricacy and the minuteness of numerous communications between spaces and the delicacy of the trabeculae which separate air cells from marrow spaces render impossible the absolute identification of the two kinds of spaces. Furthermore, the corrosion method is undependable, since the material employed may spread only to the air cells immediately adjacent to the larger cavities. The deep and highly protected position of the petrous portion of the temporal bone and the absence of any constant pattern of pneumatization serve to reduce greatly the dependability of roentgenograms for anatomic study of the air cells. Without question, the microscopic examination of serial sections of temporal bones is the only method which permits of accurate identification of pneumatic and diploetic spaces in the bone. The serious shortcoming of this method, however, is the difficulty of visualizing in three dimensions the form and the relations of the cavities and of their communications seen microscopically in only two dimensions and in a single section at a time.

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3. Belinoff, F., and Balan, M.: Ueber den Bau der Pyramidenspitze, Monatschr. f. Ohrenh. **64**:1185-1188, 1930.

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6. Myerson, M. C.; Rubin, H. R., and Gilbert, J. G.: Anatomic Studies of the Petrous Portion of the Temporal Bone, Arch. Otolaryng. **20**:195-210 (Aug.) 1934.

7. Friesner, I.; Druss, J. G.; Rosenwasser, H., and Rosen, S.: Suppuration of the Petrous Pyramid, Arch. Otolaryng. **22**:659-688 (Dec.) 1935.

8. Jones, M. F.: Symposium on Certain Fundamentals in Regard to Suppuration of the Petrosal Pyramid: Anatomy, Arch. Otolaryng. **22**:515-516 (Oct.) 1935.

MATERIALS AND METHODS

For the present study we employed the only method which has none of the limitations described, namely, the wax plate method of reconstruction,[9] utilizing it for the first time in the study of the air cells of the petrous portion of the temporal bone.[10]

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9. Two reconstructions were prepared of the air cells and related structures at a magnification of 25 diameters. They represent somewhat more than that part of the series of sections which includes the cochlea. One reconstruction (figs. 20 to 22) represents bony tissue only, and therefore the trabecular walls of the air spaces and the marrow spaces are shown, as well as the compact bone of the otic capsule. In this reconstruction bone is treated as if it were solid, i. e., without taking into account the haversian spaces. The reconstruction was fabricated in seven pieces, the levels being selected on account of their topographic importance. Each piece includes the craniocaudal extent of approximately fifty sections, i. e., of 1.25 mm., in the actual bone. The pieces portray, as sections notably fail to do, the course of such channels as the carotid canal, the meatus and the labyrinth. The other reconstruction (figs. 2 to 9) represents not the osseous wall of the spaces but the spaces themselves, enclosed by mucous membrane (tympanic cavity, auditory tube, air cells) or by bone (meatus, osseous labyrinth, canals for vessels and nerves, diploetic spaces for marrow). On the reconstructions (figs. 2 and 3) the levels of the sections are indicated; the actual serial number is employed, in order that one may compute the thickness of any "slice" of the bone as represented by a portion of the reconstruction, or the distance between levels. Since the sections are 25 microns thick, the distance between sections 662 and 324, for example, is 8,750 microns, or 8.75 mm.

Our experience in demonstrating this reconstruction before clinical groups has revealed an unfamiliarity with the details of the process involved. A description of the procedure might therefore be of some interest. Tracings were made of individual sections by means of the Edinger projection apparatus at a magnification of 25 diameters. The tracings resemble the drawings seen in figures 10 to 19. These tracings were then transferred to wax plates of correct thickness. Cutting the wax plates according to the pattern of the tracing gave a duplicate of each original section, magnified twenty-five times in all dimensions. By piling these wax plates in proper serial sequence, a three dimensional reconstruction was produced, in which anatomic features and relationships were reproduced in correct positions. By this method proper preservation and preparation of tissues are assured; sections are available for future reference and comparison; histologic identification of air cells and marrow tissue is secured, and structures seen microscopically in two dimensions (in the sections) are reproduced in three dimensions and gross size.

The drawings of the sections (figs. 10 to 19) were prepared from tracings made with a projection apparatus at a magnification of 12.5 diameters. One of the group of nine drawings includes the membranous labyrinth, nerves and mucous membrane (fig. 17); the others exclude these soft tissues, showing only the osseous structures (figs. 10 to 16, 18 and 19), as does the sectional reconstruction (figs. 20 to 22).

10. J. S. Fraser and J. K. Milne (Reconstruction Model of the Right Middle and Inner Ear, J. Anat. & Physiol. **49**:119-135, 1915) made a reconstruction of the principal cavities in the temporal bone by a similar method for classroom demonstration. In their report they described numerous small cells, but their illustrations do not indicate that any such cells were incorporated in their reconstruction.

The specimen selected was removed at autopsy from the right temporal bone of a child 4½ years of age.[11] The entire petrous portion and part of the mastoid were included in the excised block. The block was immediately fixed with solution of formaldehyde, decalcified in a dilute alcoholic solution of nitric acid and embedded in pyroxylin. Sections were cut at a thickness of 25 microns and were stained in hematoxylin and eosin; they include the entire depth of the bone, and are 800 in number. All technical procedure was carried out by Miss Helen Banks, R.N. The Children's Memorial Hospital supplied the specimen.

OBSERVATIONS AND COMMENT

The petrous portion of the temporal bone is roughly pyramidal, with the apex pointing inward and forward; it is wedged in between the basioccipitalis and the great wing of the sphenoid bone, where it forms a part of the floor of the cranium (fig. 1). Its superior angle is raised to form a prominence which separates the middle from the posterior fossa of the cranial cavity (fig. 1; compare figs. 10 and 20). The entire petrous part is so situated in the skull that its long axis passes in an oblique plane from the apex in an anteromedial direction to the epitympanic portion in a posterolateral direction (fig. 1*a*). The various structures within the bone with which we are at present concerned (figs. 1 and 1*a*) are the air cells and the marrow spaces at the petrous apex; the carotid canal, to which pneumatic and diploetic tissues are closely related; the cochlea and the internal meatus; the semicircular canals; the air cells which surround the canals, and the auditory (eustachian) tube and the tympanic cavity (middle ear), which together form a continuous cavity occupying the anterior angle of the pyramid throughout its length.

The presence of cells in the petrous pyramid has long been recognized, and tubal cells have frequently been described, but their form as an assemblage and their exact relation to the parent tympanic cavity have never been satisfactorily established. In microscopic study of serial sections the relationships of the air cells appear hopelessly confusing. But when viewed entire they are found to assume a fundamentally simple pattern, related chiefly to the carotid canal; the cells are outgrowths of the posteromedial wall of the tympanic cavity near the orifice of the auditory tube. As if prevented by the presence of the carotid canal from continuing directly inward from this area of tympanic communication (fig. 7), evaginations seem to have extended around the canal in every possible direction. Some air cells extend

11. This is the first in a projected series of such studies. A report on the air cells of a 13 year old child and of a young adult (18 years old) is in preparation. It is our plan to include, ultimately, representatives of the various age groups.

upward and backward around the carotid canal, pneumatizing the area of bone between the arch of the canal and the cochlea (figs. 4 to 7); others extend along the canal, then dip downward and beneath its loop and finally attain its internal aspect, the cells together producing an almost continuous circle of pneumatized tissue. The cells as a whole, with their tympanic roots, may be described as assuming the form of a tree (see especially fig. 9), a smaller set stretching horizontally and then upward from beneath the arch, and the terminal branches of the two sets just failing to touch each other (figs. 5 and 7).

Since they are disposed in pericarotid fashion, the groups of cells may be described on the basis of their relation to the carotid canal. There are four such groups, which, for convenience, will be designated A, B, C and D (figs. 2 to 9). Of course, all adjacent cells intercommunicate freely. They are irregular in form and anastomose through relatively large openings. Small cells of group *C* are from 1.5 to 2 mm. long and from 1 to 1.2 mm. high; large cells of the same group (at the tip) measure 4 mm. in length and 3 mm. in height. The largest of group D are 6 mm. in length and 4 mm. in height.

An unbroken wall of bone everywhere separates the air cells from the marrow. At no point is there evidence of epithelial encroachment on the marrow spaces, and it would therefore appear that a basically stable pattern has been established.

With this picture of the general pattern of pneumatization in mind, one can study the relationships of the groups of cells to the various structures seen in individual sections at illustrative levels in the reconstructions.

Figure 10, section 620 (see level in figs 2 and 3), topography: The section includes the petrous portion of the temporal bone from its apex (at the left) to its base (at the right). The enlarged portion at the right is the arcuate eminence, and within the bone at this level is the superior semicircular canal, cut near its upper limit.

Apical cells (blue) of group B occur on the anterolateral surface of the bone, or, more precisely, in the wall of the middle cranial fossa; they do not communicate with the epitympanic cells of the mastoid area, being separated from them by the solid tissue of the otic capsule. Marrow (red) here occupies almost the entire apex; 1 mm. above the level represented by this section it actually does occupy all of it (see level of section 669 in fig. 20).

The air cells in the lateral portion are inward extensions of epitympanic cells, but the dense otic capsule presents an effective barrier to their further extension medialward. They do not communicate at this or at any other level with the apical cells. The thinness of the bony wall at the petrous tip of this end of succeeding sections is an arresting structural feature.

Figure 11, section 570, topography: The section passes through the roof of the internal acoustic meatus, the open cranial portion of the carotid canal and the superior semicircular canal, lodged in the compact bone of the otic capsule.

The apical air cells here occupy the entire anterolateral wall and are separated from the mastoid cells by the solid bone of the otic capsule, which overlies the cochlea and the vestibule. The epitympanic cells penetrate the bony tissue enclosed in the loop of the semicircular canal, almost completely surrounding the lateral extremity thereof; they also lie on both sides of the subarcuate fossa.

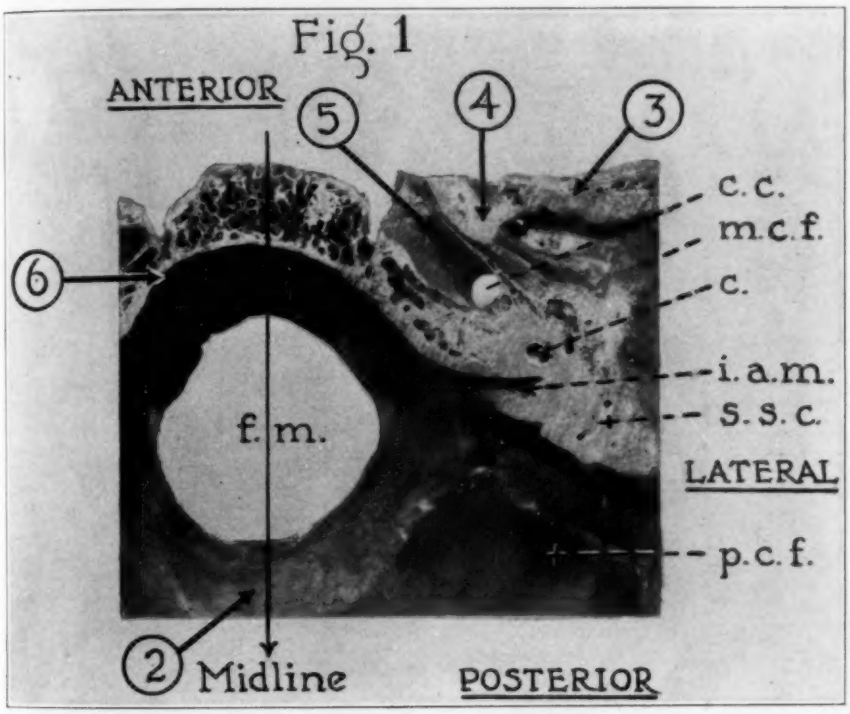


Fig. 1.—The floor of the cranial cavity (posterior and middle fossae), cut parasagittally to the left side of the foramen magnum: petrous portion of the temporal bone ground to the level of the carotid canal, basal turn of the cochlea and internal acoustic meatus. The level is near that of figures 1*a* and 12. The horizontal direction of each of the views seen in figures 2 to 6 is indicated by correspondingly labeled arrows.

In this and in the succeeding figures *a.c.* indicates air cell; *a.t.*, auditory tube; *c.* or *coch.*, cochlea; *c.c.*, carotid canal; *e.a.m.*, external acoustic meatus; *e.r.*, epitympanic recess; *f.c.*, facial canal; *f.m.*, foramen magnum; *i.a.m.*, internal acoustic meatus; *j.f.*, jugular foramen; *m.*, marrow; *m.a.c.*, mastoid air cells; *m.c.f.*, middle cranial fossa; *o.t.c.*, orifice of tympanic communication; *p.c.f.*, posterior cranial fossa; *s.s.c.*, superior semicircular canal; *t.c.*, tympanic cavity; *t.m.*, tympanic membrane, and *v.*, vestibule.

The apical air cells of group B appear broader than in the preceding figure, since the section passes through them as they arch over the carotid canal (figs. 4 to 8); actually they lie within its superior wall (compare the following section, fig. 12) and are separated from the dura mater of the middle cranial fossa merely by the thin lamina of bone which constitutes the inner table of the skull.

An unbroken bony septum completely separates the air cells from the adjacent marrow spaces. The upper portion of the labyrinthine

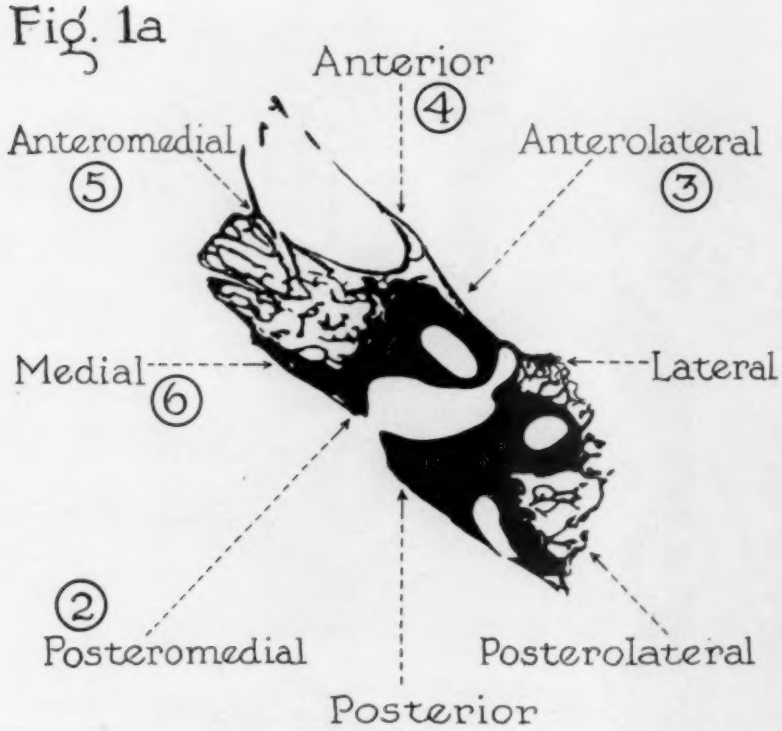
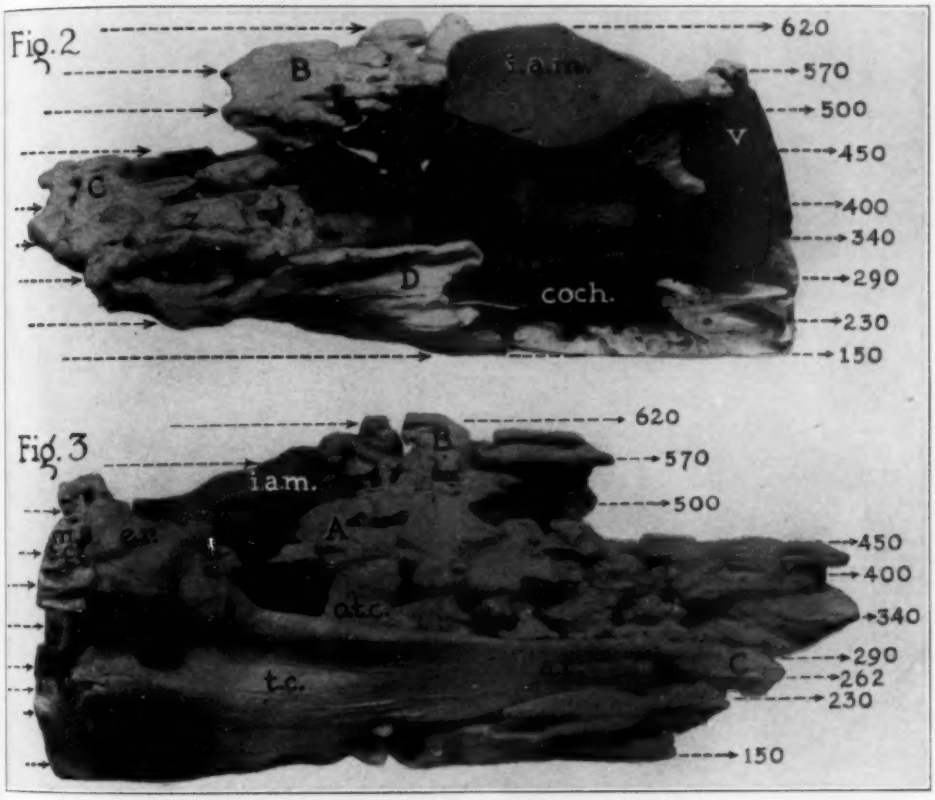


Fig. 1*a*.—Section 500 (shown in fig. 12), near the horizontal level of the ground surface portrayed in figure 1, placed in a corresponding position, with the surfaces marked and the direction of the views seen in figures 2 to 6 indicated by correspondingly numbered arrows.

capsule, invaded at this level by the internal acoustic meatus, intervenes between the antelabyrinthine, or apical, air cells and the postlabyrinthine, or epitympanic, cells. While our present study is concerned primarily with the area medial to the labyrinth, certain important relationships in the postlabyrinthine area are so clearly portrayed in this section that they seem to warrant brief comment. In the literature one frequently finds reference to "chains" of mastoid cells and to "fistulous tracts"; following such a chain, by probing, the operator has been guided

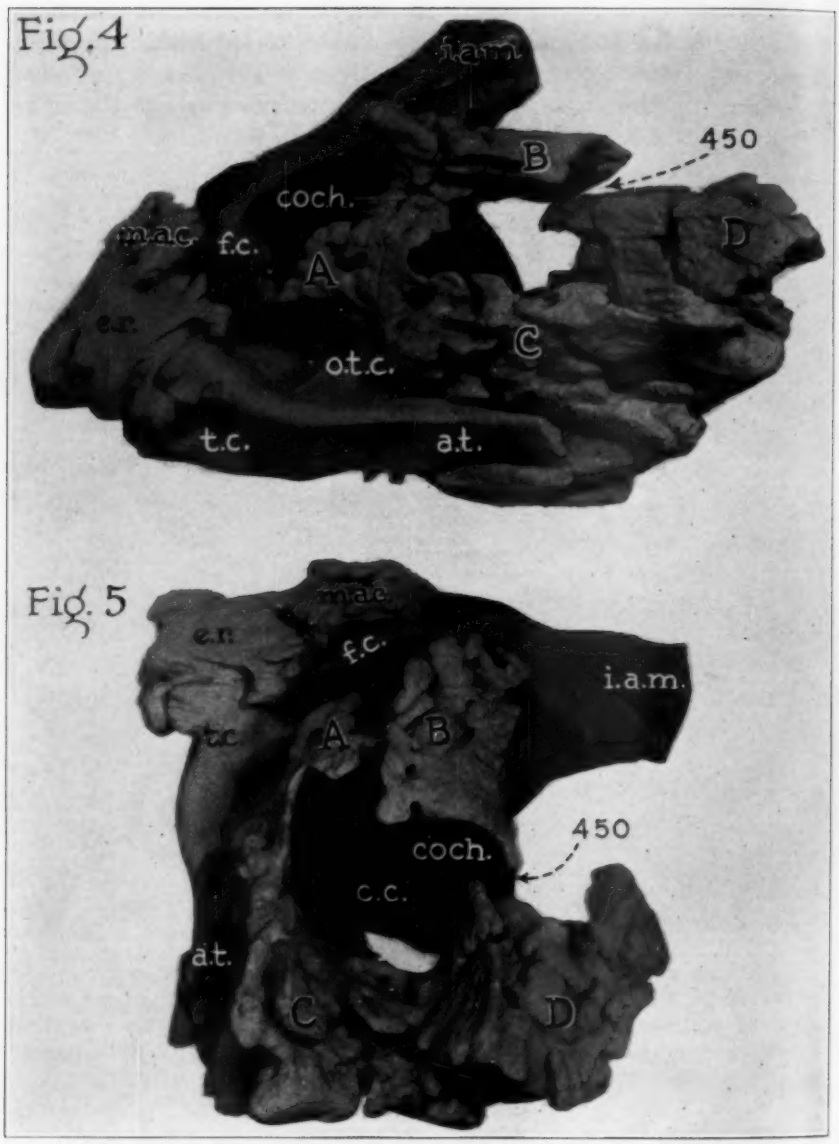
to infected areas situated deep within the pyramid. There is such a chain of cells in our specimen lateral to the superior semicircular canal, but it does not extend deeply within the pyramid. Rather, it proceeds as far as the labyrinthine capsule, where it contributes to the formation of a pneumatic ring about the anterior orifice of the superior canal. Equally significant is the relation of this chain of cells to the subarcuate fossa. The fossa penetrates the area deeply and is bounded by air cells; it is



Figs. 2 and 3.—Reconstruction of air cells and related structures of the petrous portion of the right temporal bone. Magnification 4½ diameters. Figure 2 presents the posteromedial view. The levels of the sections are shown by lines correspondingly numbered, and the groups of air cells are labeled to correspond with the designations in figures 10 to 19. Figure 3 presents the anterolateral view.

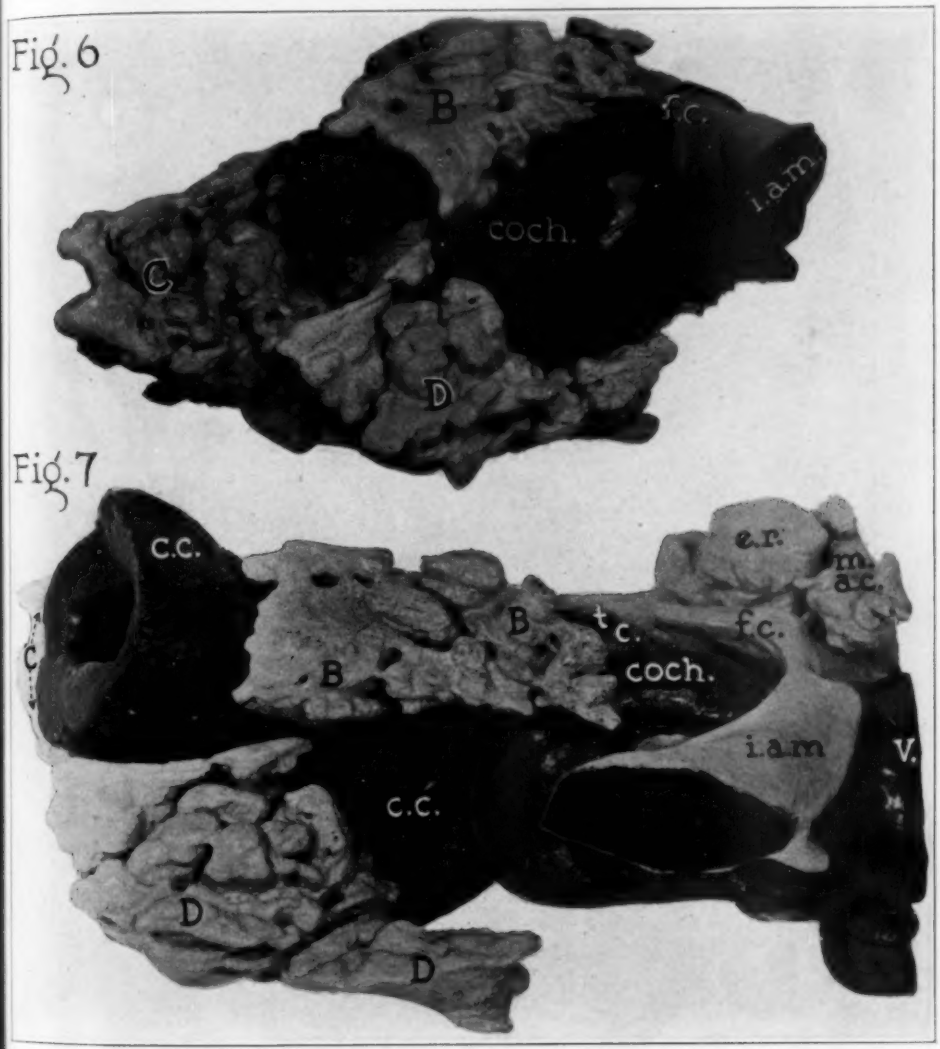
itself a pathway to the meninges, and it is one of the routes named by Perkins [12] along which petrous infections may reach these membranes. The danger of probing such a chain of cells is obvious.

12. Perkins, C. E.: Abducens Paralysis and Otitis Media Purulenta, Ann. Otol., Rhin. & Laryng. **19**:692-703, 1910.

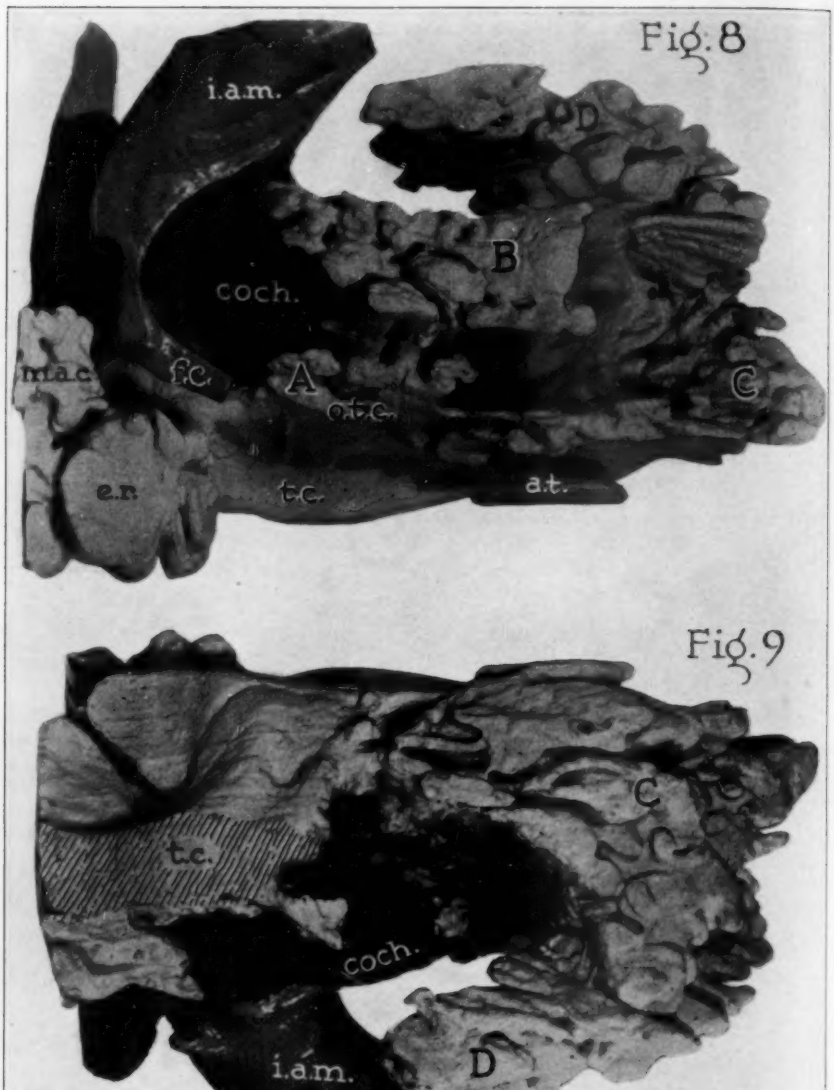


Figs. 4 and 5.—The reconstruction, continued. Magnification 5 diameters. Figure 4 presents an anterior and somewhat superior view, showing especially the origin of the air cells from the tympanic cavity, and figure 5, an anteromedial and somewhat superior view, showing the cells of groups B and D which fail to meet (at the arrow marking the level of the section in figure 13).

Figure 12, section 500, topography: The section passes through the foramen lacerum, i. e., the cranial opening of the carotid canal (not a complete canal in the two preceding sections), through the superior portion of the cochlea, through the facial canal in its lateral course, above the vestibule at the level of the genu



Figs. 6 and 7.—The reconstruction, continued. Figure 6 presents a medial and somewhat superior view. Magnification 5 diameters. The cells of the superior and the inferior group are seen in the foreground. Figure 7 presents the postero-medial view. The representation of the carotid artery is included (removed in figure 2 to expose the cells of group C).



Figs. 8 and 9.—Reconstruction. Magnification 5 diameters. Figure 8 presents the superior view and figure 9 the inferior view.

of the facial nerve and through the internal acoustic meatus. The crura of the superior semicircular canal are approaching their points of origin from the vestibule. The upper portion of the basal turn of the cochlea comes into view in this section, lodged in the solid bone of the labyrinthine capsule.

At this level the apical cells of group B ascend in the anterolateral wall of the carotid canal; they spread over this wall to attain its posteromedial aspect. They are near, but are not continuous with, the epitympanic cells which surround the semicircular canal. On the posterolateral aspect of the carotid canal may be seen the constricted portion of the supracarotid group of air cells (group B). Having overarched the canal, the cells now appear on both aspects thereof (compare figs. 2, 6 and 7).

The cells thus far discussed belong to the supracarotid group, which constitutes the smaller branch of the "tree." The main bulk of this entire group has dural relationships both before and behind, a circumstance which makes the thinness of the anterior wall of the air cells highly significant.

Figure 13, section 450 (compare fig. 21), topography: The section is superior to the cavity of the auditory (eustachian) tube, passing through its superior wall; it includes, however, the space of the tympanic cavity (middle ear). It also passes through the basal and central coils of the cochlea, the superior vestibular area, the superior portion of the spiral foraminous tract and the descending portion of the facial (fallopian) canal. The septum across the tympanic cavity is the downward projection of the roof which separates the tympanic recess from the mastoid antrum (arrow at upper right of figure).

Air cells extend along the anterolateral aspect of the carotid canal from the apex of the cochlea toward the tip of the pyramid; marrow occupies the tip of the bone and the posteromedial wall of the canal. The apical air cells are those of groups A and C (figs. 3 and 4). The former cell group lies just lateral to the cochlea, near its apex; the latter is situated in the anterolateral wall of the carotid canal. It is to be noted that the supracarotid cells, group B (fig. 12), are not represented at this level and that the infracarotid cells, group D, present at a more inferior level (fig. 15), have not yet appeared. This means that the present section passes through the area between these groups (see arrows, figs. 4 and 5).

Figure 14, section 400, topography: The section passes through the facial canal in its descending course, the modiolus of cochlea, the vestibule and the floor (at its lateral end) of the internal acoustic meatus. The level is below that of the floor of the cranial cavity, so that the great wing of the sphenoid bone in relationship with the anterior aspect of the petrous apex forms the semicanal for the tensor tympanic muscle. The carotid canal is more deeply set within the apex; the dense bone of the labyrinthine capsule forms part of its posterior wall.

The apical cells now extend beneath the carotid canal, as the groups designated C and D (figs. 5 to 7); the continuity of these groups, here separated by the carotid canal, is evident in the next level illustrated (fig. 15). They cover four fifths of the circumference of the canal; pressing into the very tip of the petrous portion, they replace the marrow, which was abundant at more superior levels (figs. 11 and 12). Here, as elsewhere, the bony wall which separates air cells from marrow is of a thickness equal to that of the carotid canal or of the inner lamina of the skull.

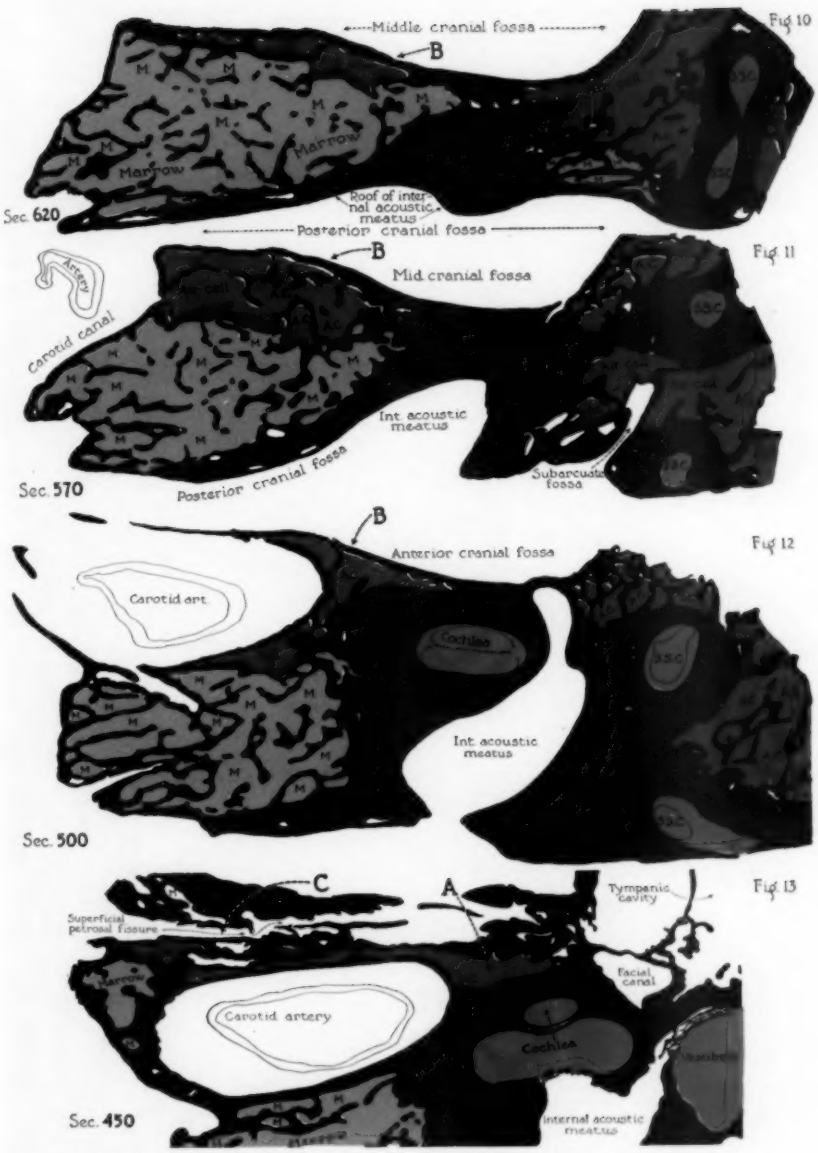
Figure 15, section 340, topography: The section passes through the vestibular (oval) window, the three ossicles, the auditory tube and the inferior portion of the semicanal for the tensor tympani muscle. The carotid is still more deeply set and is here cut in its most perpendicular portion; its relationship to the otic capsule is such that a portion of the latter still forms a quadrant of the wall of the canal. The section is inferior to the internal acoustic meatus, passing through the lower fourth of the cochlea.

At this level cells are at their maximal extent; marrow is confined to a small medial area. The air cells have as yet no communication with the middle ear. Group A extends toward and lies on the lateral aspect of the cochlea, and group C occupies the anterolateral wall of the carotid canal; the latter group then crosses beneath the canal as group D, attaining the posteromedial wall (see especially figs. 5 to 7).

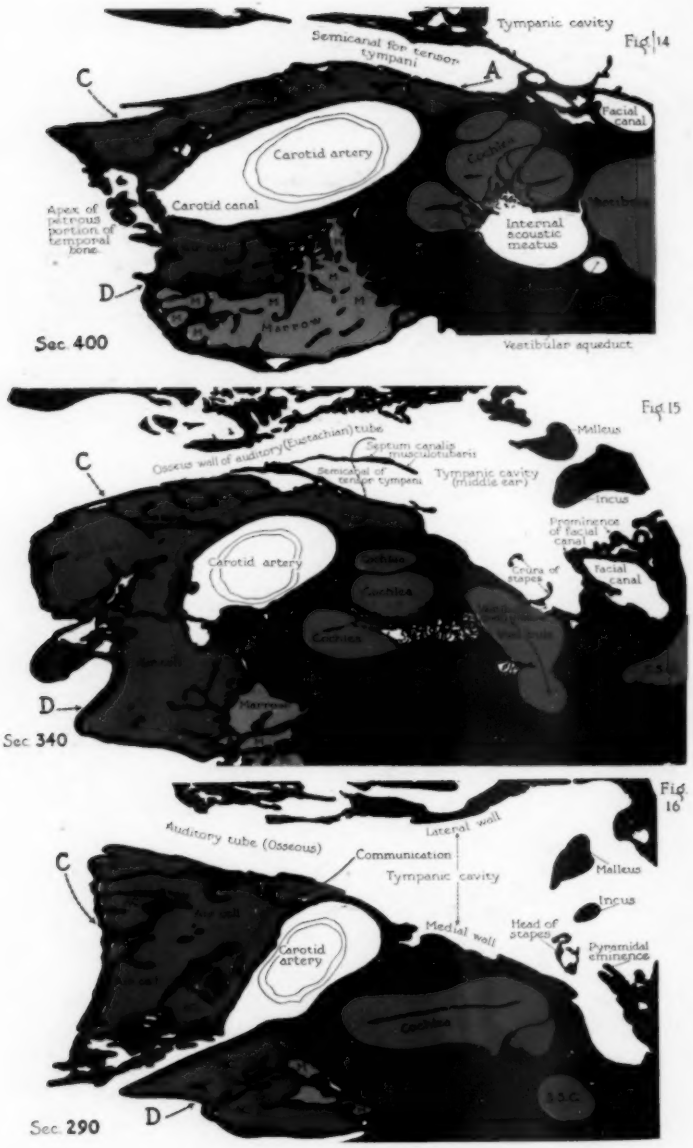
The greatly expanded walls of the carotid canal, through three fourths of the circumference, are pneumatized with large air cells. These cells are infracarotid; that is, they lie within the arc described by the carotid in its course through the apex (fig. 7). In this section the displacement of the marrow spaces by pneumatic cells is more striking than at any level yet examined. The proximity of the most laterally placed air cell to the apex of the cochlea is noteworthy. At no point in our reconstruction do we find any air cell coming closer to any portion of the labyrinth.

It is at this level that the common trunk of the treelike pattern of cells is encountered, i. e., the area near the communication with the parent tympanic cavity (compare figs. 3 and 4). This common root is a downward prolongation of the air cells located immediately anterior to the apex of the cochlea (at the point of the arrow in fig. 15). This communication is situated within the tympanic cavity at the orifice of the auditory tube, immediately beneath and obscured by the bony shelf (processus cochleariformis) of the semicanal for the tensor tympani muscle (compare figs. 16 and 17).

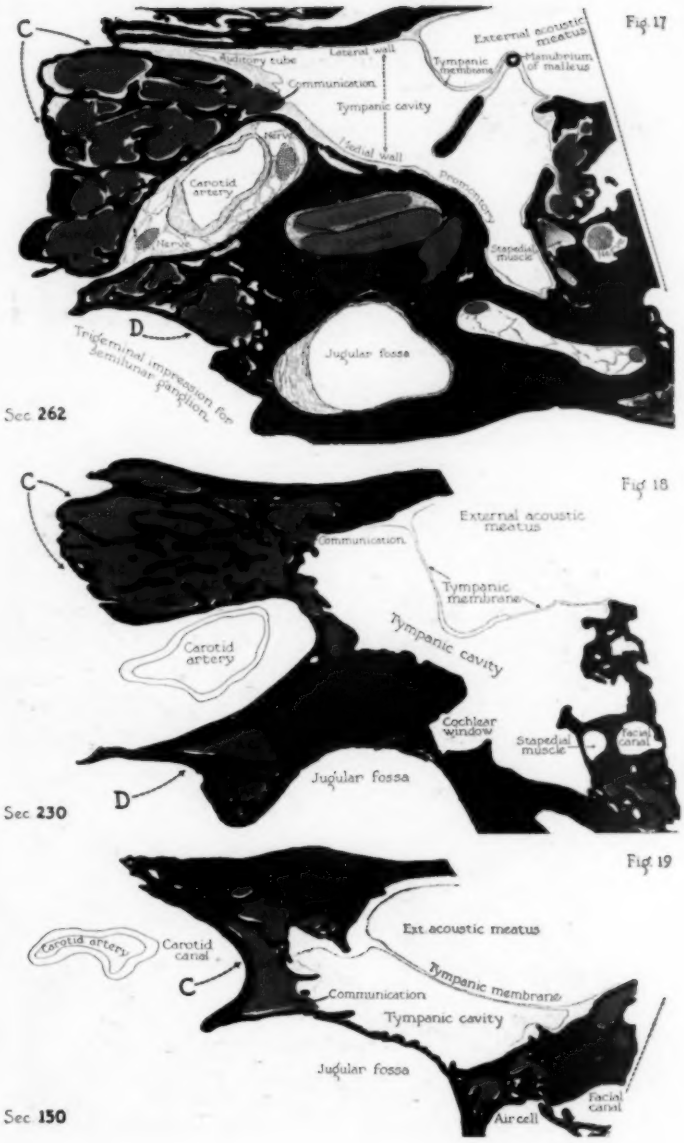
Figure 16, section 290, topography: The section is situated just below the completely closed portion of the carotid canal, near the lower surface of the petrous portion of the temporal bone; it is below the level of the vestibular



Figs. 10 to 13.—Sections of the petrous portion of the temporal bone: marrow, red; air cells, blue, and membrane of the labyrinth, purple. Magnification 3.3 diameters.



Figs. 14 to 16.—Sections, continued. Magnification 3.3 diameters.



Figs. 17 to 19.—Sections, continued. Magnification 3.3 diameters.

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window and passes through the pyramidal eminence for the stapedius muscle, the lower portion of the basal turn of the cochlea and the head of the stapes.

The abundant air cells of the petrous tip (groups C and D) are separated at this level by the opening of the carotid canal, thus assuming precarotid and postcarotid positions. Here occurs the topmost limit of the communication of the tympanic cavity and the apical air cells (see also fig. 17); it is situated just anterior to the prominence produced on the medial tympanic wall by the bulging carotid canal. Marrow tissue has been almost entirely replaced by air cells. It should be noted that the cells present in section 340 (fig. 15) on the anterolateral aspect of the cochlea are here wanting and that the tympanic cavity is correspondingly wider; the significance of this change is evident when figures 15 and 16 are compared.

Figure 17, section 262, topography: Nearing the inferior limit of the auditory tube, the section passes through the tympanic membrane with the attached manubrium of the malleus, through the promontory and through the summit of the jugular fossa. The carotid canal here opens inferiorly on the base of the skull. The section also includes the lower portion of the basal coil of the cochlea, the posterior semicircular canal, the facial nerve within the lateral wall of the tympanic cavity en route to the stylomastoid foramen, and the constricted channel through which the tympanic cavity communicates with the cochlear (round) window. Here within a single section are exposed to the tympanic cavity the following important structures: the facial canal, the stapedius muscle, the posterior semicircular canal, the cochlea and the carotid canal, in that succession from posterior to anterior.

Air cells belonging to groups C and D occupy the entire tip, marrow being totally absent; cells of group C open into the tympanic cavity at its point of junction with the auditory tube.

From an inferior aspect, a recess is evident in the medial wall below the line of attachment of the processus cochleariformis; throughout the length of this sulcus is seen the aditus of the communication. This communication leads upward and inward into the groups of cells previously described as groups A and C. This primary communication assumes the character of a longitudinal, slitlike aperture, extending from the summit of the promontory to that of the prominence produced by the carotid canal, a distance of almost 7 mm. This aperture is narrow at each end and widens to its greatest diameter (approximately 1 mm.) in the excavation between these two prominences. By way of comparison, the greatest diameter of the cochlear window is not more than 1 mm., while the greatest dimension of the vestibular window is approximately 2.5 mm. The importance of a natural communication of such a size at this particular point is obvious; it may serve as an avenue for extension of an infection or as the route for spontaneous or artificial drainage.

While the failure of earlier investigators to recognize this point of direct communication with the tympanic cavity may be charged to the cursory nature of routine laboratory and clinical examination, numerous reports mention the occurrence in this territory of "fistulas" seen at operation or at autopsy. Thus, Myerson, Gilbert and Rubin [6] reported from a study of the literature 53 cases in which fistulas had been discovered, and they observed that in 18 cases of their own fistulas "originated in or about the orifice of the eustachian tube." Kopetzky and Almour [13] were impressed by the occurrence of "fistulous tracts" at the same point, which "indicated that pus within the petrous apex usually finds spontaneous egress through the peritubal cells." In a case of Gradenigo's syndrome, Mayer [14] made a wide exposure of the floor of the middle ear, through which granulation tissue protruded. After a probe was introduced and this "hole" widened with a curet, a profuse amount of pus escaped, and the patient, thus relieved, recovered completely.

Air cells which are extensions of the epitympanic group communicate with the tympanic cavity near the point of attachment of the tympanic membrane.

The manner in which the mucoperiosteum bridges intervals in the bony trabeculae which separate individual air cells is evident, as is also the extent to which it projects from the bony margin of the tympanic aditus as a prominent lip.

Figure 18, section 230 (compare fig. 22), topography: The section passes through the widely open inferior portion of the carotid canal, the lower reaches of the posterior semicircular canal, the cochlear (round) window, the jugular fossa and the tympanic membrane inferior to the manubrium of the malleus. The second section described (570, fig. 11) represents a level above the osseous portion of the carotid canal; the present section lies below the level of the intraosseous portion of the carotid.

Apical cells of group C are still numerous at this level; a few of group D occur in the spur of bone separating the carotid canal from the jugular fossa. The latter cells communicate with the former at the higher level (section 340, fig. 15) and, by way of the cells of group C as intermediaries, with the tympanic cavity (figs. 3, 4 and 8). One of the multiple small communications occurs here, entering a proximal air cell directly from the tympanic cavity.

Figure 19, section 150, topography: The section is near the floor (jugular wall) of the tympanic cavity. It passes through the facial canal near its exit

13. Kopetzky, S. J., and Almour, R.: Suppuration of the Petrous Pyramid: Pathology, Symptomatology and Surgical Treatment, Ann. Otol., Rhin. & Laryng. **39**:996-1016, 1930; **40**:157-177, 1931.

14. Mayer, O.: Gradenigoscher Symptomkomplex bei akuter Mittelohreiterung. Monatschr. f. Ohrenh. **63**:970-974, 1929.

from the stylomastoid foramen, through the inferior portion of the tympanic membrane and through the widely open jugular fossa. The close relation of the jugular fossa to the tympanic cavity is a striking feature; only an extremely thin plate of bone intervenes between the two spaces.

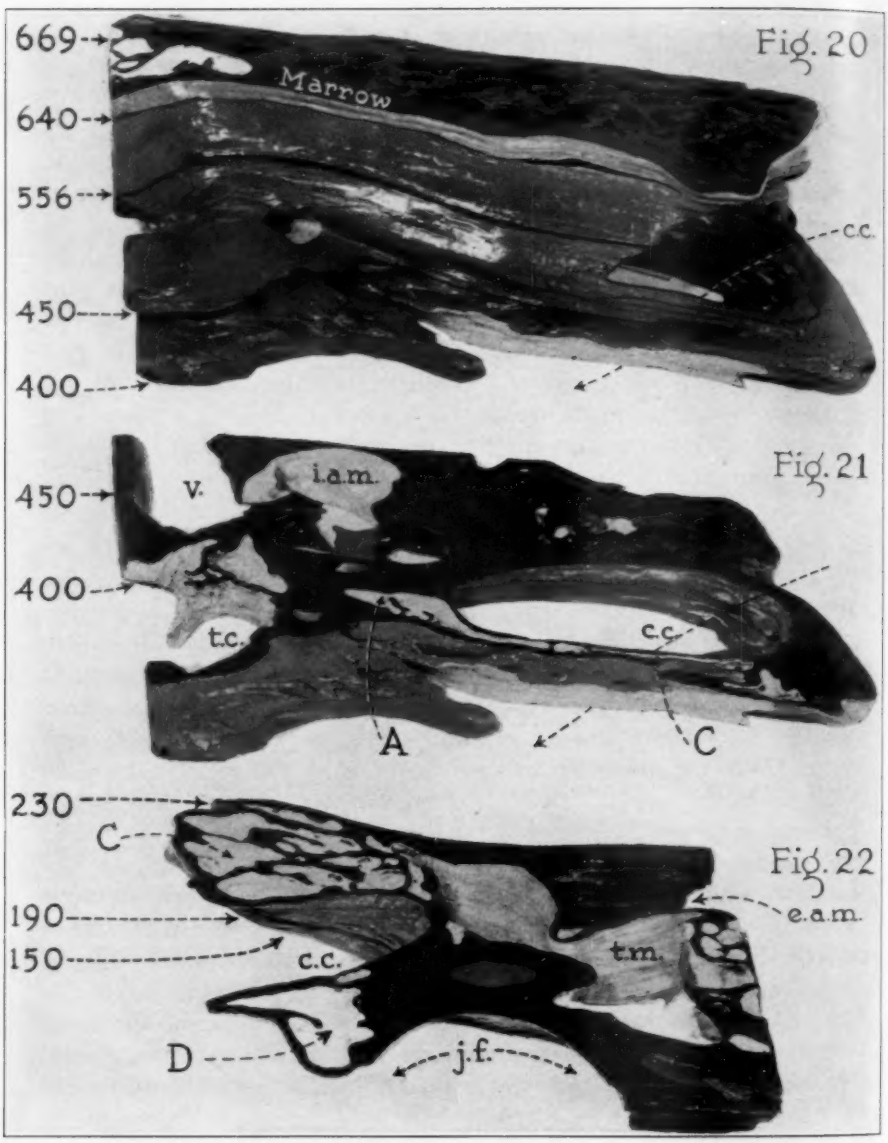
Here, close to their inferior limit, the precarotid air cells still open into the middle ear, by a communication comparable to a nasal orifice of an ethmoid cell. This is the lowermost tympanic communication of the cells in the petrous tip. The dimensions of this aperture are 1 mm. by 0.5 mm.; it opens medially into the pericarotid group of cells designated as group C. Three other apertures, two slightly larger than the one just described and the third only about half as large, are found just within the tympanic cavity, along the line of the attachment of the tympanic membrane to the lateral wall. They are inferiorly placed and open into the lowermost of the cells, designated groups C and D (compare Siebenmann,[1b] figs. 18, 19).

CONCLUSIONS

In our specimen of the temporal bone of the child there is extensive pneumatization of the petrous portion. Pneumatization has apparently reached its basic pattern, since, although marrow spaces lie adjacent to air cells, a bony wall everywhere separates the two. This partition contains only minute hiatuses, through which blood vessels pass between the vascular marrow and the substrate of the mucous membrane; there is no histologic indication of encroachment of air cells on marrow. Assumedly, only such gradual architectural alterations will subsequently occur as are regular features of the life history of any bony element in the human skeleton.

Pneumatic spaces almost completely fill the tip in its inferior three fourths, the superior fourth being occupied by marrow; the latter is chiefly dural in its cranial relations and the former tympanic, carotid and jugular.

Since in this study we have based our descriptions on wax plate reconstructions made to scale from serial sections of the temporal bone, we are able to demonstrate not only the form of individual cells and of the mass of pneumatized tissue as a whole but the relations of the assemblage of cells to structures within the temporal bone which are important anatomically and surgically. The importance of these relationships is increased by the excessive thinness of the bony walls of the air cells. The cells which occupy the petrous tip are extensions of the tympanic space, near the area of continuity of the tympanic cavity with the auditory tube. In this zone of origin the proximal cells lie anterolateral to the carotid artery, occupying virtually the entire thickness of the wall of the carotid canal. This relationship to the canal is



Figs. 20 to 22.—Reconstruction of air cells, marrow spaces and related structures. The levels are indicated; compare figures 10 to 19. Magnification 4½ diameters. Figure 20 presents the anterolateral view of the section superior to that represented in figure 10 and shows the superior surface. Figure 21 presents the anterolateral view of the reconstruction of the fourth piece and shows the upper surface at the level of the section represented in figure 13. Figure 22 presents the posteromedial view of the reconstruction of the sixth and seventh pieces and shows the upper surface near the level of the section represented in figure 18.

fundamental, even the most distally placed cells not departing far from the carotid territory. The cells are disposed in pericarotid manner, forming an almost complete circle of pneumatized tissue. From the origin, the group of cells extends a short distance posterolaterally, as far as the apical turn of the cochlea. Another and larger group extends upward, spreading over the arch of the carotid canal and then downward on the posteromedial aspect thereof; these cells are separated from the subdural bone of the cranial fossa by a shell of marrow tissue. As it descends, the group becomes both wider and longer. From the same tympanic origin a third group extends forward, i. e., anteromedially, along the course of the auditory tube. Dipping downward beneath the loop of the carotid canal, the cells lie in the walls of the jugular and the mandibular fossae. Those which ascend in the medial wall of the carotid canal approach, but do not communicate with, the cells which overarch the canal. The most posterior members of the group extend almost as far as the basal turn of the cochlea and the internal acoustic meatus but not into infralabyrinthine or supralabyrinthine territory.

The pericarotid position of the apical air cells and their continuity with the tympanic cavity in a small area suggest that pneumatization of the antelabyrinthine area represents an extension of mucous membrane from the tympanic wall and that, encountering the carotid artery as an obstruction, it becomes arboriform, within the bony wall of the carotid canal, forming an annular collection of cells. Extension of cells occurs as far as the very tip of the bone in one direction and to the cochlea in the opposite direction. The communications are assumedly the fistulas seen by otolaryngologists in operations for suppurative conditions.

In our specimen there is no direct communication across the labyrinthine bone between the apical air cells and those of the epitympanic and mastoid portions of the temporal bone; the solid bone of the otic capsule seems to have provided an effective barrier against posterior extension of the apical cells. The cells of the epitympanic group penetrate the bone of the canalicular area, almost surround the superior semicircular canal and lie on both sides of the subarcuate fossa; yet they are not contiguous to the most posterior of the apical cells. No anastomosis occurs between any pneumatic spaces in the apex and similar spaces in the sphenoid and occipital bones.

Some of the apical cells are related to the auditory tube, but only by position, not by origin. No cells arise from the tube itself; they merely extend along the tube from an origin on the tympanic wall.

TREATMENT OF CHRONIC CATARRHAL DEAFNESS WITH THE EUSTACHIAN HEAT BOUGIE

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I have been using the eustachian heat bougie for treatment of various conditions of the eustachian tube and middle ear for over five years. Regularly I have received communications inquiring as to the results obtained with the heat bougie, which I presented[1] three years ago.

The eustachian heat bougie is used with the ordinary eustachian catheter; it comes in graduated sizes, the smallest being 2.5 to 3 French in diameter, that is, about the size of a no. 2 whalebone bougie. The bougie consists internally of two separate, well insulated circuits: (1) a fine resistance wire through which a low grade current runs from a control rheostat, furnishing any desired heat up to about 180 F., and (2) a circuit with a thermocouple in the distal portion of the bougie, which serves to register continuously the temperature of the mucosa with which it is in contact.

In the previous article I wrote: "As to the therapeutic value of the heat bougie compared to other methods, I think the heat bougie will open the most rigid stricture, will keep the tube open longer with less frequent treatment than any other procedure mentioned." At present, after continued experience, I am of the same opinion. A palpable stricture that one feels on entering the tube cannot be felt when one withdraws the bougie. After a few treatments the stricture cannot be discerned, although it has returned in some cases after several months. However, the actual clinical value of the heat bougie in terms of improved hearing, diminished tinnitus and lessened fulness in the ear is a variable. Early in my experience with the bougie I found that some patients were helped by its use: some with regard to hearing, fulness and tinnitus and others with regard to hearing and fulness only, tinnitus remaining the same. In all cases I was able to open the eustachian tube. Unfortunately, opening the eustachian tube helped only certain patients, those who as far as hearing was concerned had no fixation of the stapes.

It was stated in the first paper:

> If the inflammatory process is limited to the tube it will certainly improve the hearing and tinnitus, and this is the result we have obtained in such cases. If the

1. Simon, E.: A New Treatment for Eustachian Tube Obstruction: Controlled Heat Bougie, Ann. Otol., Rhin. & Laryng. **43**:598 (June) 1934.

process has gone on into the middle ear it will help the patient if there are no adhesions and fibrosis. However, if there are scar tissue and adhesions there usually is some involvement of the stapes, often with some fixation. We have definitely improved hearing in some cases with a fixed adherent drum, but in general the outlook is unfavorable.

If there is fixation of the stapes, neither treatment of the eustachian tube nor any other form of treatment to my knowledge will improve hearing. Unfortunately, there is no accurate criterion other than inflation or trial bougienage for determining impaired movement of the stapes. However, if after three or four treatments the tube has been kept well open and one sees no clinical improvement in hearing, it is evident that at least one is doing no good and it is probable that one is dealing with fixation of the stapes.

A fixed or adherent drum or even fibrous tissue in the middle ear does not mean obstruction to stapedial motion and loss of hearing. McKenzie [2] wrote: "In short, otosclerosis may be associated with any kind of tympanic membrane, normal and abnormal; and so may perfect hearing. So I confess I have arrived at the stage of paying very little attention to it." However, when there is a fixed or adherent drum one must think of some adhesion in the middle ear; certainly there is a greater possibility of fixation of the stapes or of restriction of movement of the ossicular chain than when there is a nonadherent drum. Polvogt and Bordley,[3] in a thorough study of sections of temporal bones from patients with normal hearing and conductive deafness, stated:

We believe that when the Rinne test is negative it is the result of one or more lesions which interfere with the normal movement of the ossicular chain. This causes a reduction of air conduction time without changing the bone conduction time.

There is no way that I know of to determine whether any kind of treatment of the tube, that with the heat bougie in particular, will improve a patient's hearing or diminish tinnitus. The only proof is a trial: keeping the tube open for a prolonged interval, as may be done with the heat bougie. In general, a long-drawn-out course of catarrhal otitis media with a fixed drum indicates a bad prognosis. However, if a patient states that inflation of the tube gives him temporary or evanescent relief, I am pleased and promise him longer lasting improvement. I have not had to change this point of view. I do not promise

2. McKenzie, D.: Otosclerosis, J. Laryng. & Otol. **47**:92 (Feb.) 1932.

3. Polvogt, L. M., and Bordley, J. E.: Pathologic Changes in the Middle Ear of Patients with Normal Hearing and of Patients with Conductive Type of Deafness, Ann. Otol., Rhin. & Laryng. **45**:860 (Sept.) 1936.

that person a permanent cure, though I have had patients who have maintained their good hearing for several years after heat bougie treatments.

The reports given are of several recent cases selected to illustrate the value of the heat bougie, used with other treatment of the nasal sinuses.

REPORT OF CASES

CASE 1.—*History.*—Mrs. M. R., a housewife aged 38, had been very hard of hearing for the ten days before I saw her and had had tinnitus on and off during that time. One month before, she had had grip and had been in and out of bed for five days. She had had a head cold with laryngitis and a cough at that time. Her nose had been stuffed and she had had some difficulty in nasal breathing during the month preceding her first visit to me; she had had no headache except for severe occipital headache once a month (at her next visit she said that these attacks of occipital headache were more frequent). She had had a moderate number of head colds and attacks of grip. Sore throat had occurred infrequently.

Examination.—The right ear drum was slightly pale, retracted and glistening as if mucus were behind it; it was slightly movable. The left drum showed a faint pink congestion, was well retracted and glistened; it was well fixed throughout.

The nasal septum was thickened anteriorly, and it appeared to project into the right fossa and somewhat toward the left. The right inferior turbinate was large, normally pink and a trifle edematous; the posterior tip was hyperplastic. The right middle turbinate was of medium size and showed some edema. The left inferior turbinate was of medium size and was normally pink; the posterior end was pale. The left middle turbinate was of good size and was pale throughout, the paleness being more marked in the posterior two thirds. The orifices of both eustachian tubes were open. The adenoid mass was unusually large.

The right tonsil was of medium size, cryptic and slightly congested. There was a good-sized lateral pharyngeal band on the right side. The left tonsil appeared similar to the right one; there was a slightly smaller lateral band on the left side. The posterior pharyngeal wall was somewhat atrophic and had a lymphoid nodule on it 0.8 cm. in diameter.

Treatment and Course.—Routine nasal treatment was prescribed; the patient returned in eighteen days, at which time she stated that her hearing was better and that the noises in her head were less pronounced. Her ears felt more open; the left ear was slightly fuller and more "crackling." A tuning fork test, made at this visit, showed her hearing to be markedly impaired. She was given one treatment with the heat bougie and ten minutes afterward another tuning fork test.

The patient did not return for further treatment; she telephoned that she was hearing well, and that the head noises were gone.

CASE 2.—*History.*—Mr. J. H., a railroad worker aged 48, had had tinnitus on and off for the preceding two or three years. The "buzzing" was worse in the right ear and was aggravated in the winter months. Hearing was dull at times. There was crackling in both ears. The patient had had headache in the crown of his head and in the occipital region. There had been some pain in the left temporal area and slight pain in the forehead. There had been infrequent colds; the nose was stuffed and occasionally there was difficulty in nasal breathing. There

had been no sore throat. The patient had had a submucous resection five years before and had had polyps removed twice.

Examination.—The right ear drum was pale, well retracted and completely immobile. The left drum was very pale, was somewhat retracted and showed slight movement posteriorly only, when the Siegle otoscope was used.

The nasal septum was in the midline; it showed anteriorly an area of perforation about 0.6 cm. in diameter. The mucosa throughout was hypertrophic; the area of the tuberculum on the right was very much thickened. The right inferior turbinate was very large, was normally pink and showed slight edema; the posterior tip was hyperplastic. The anterior third of the right middle turbinate had been removed; the remaining portion was polypoid, with three small polyps. Some edema was present. The left inferior turbinate was large and of normal color; the posterior end was hyperplastic. The left middle turbinate had anteriorly a polyp 3 cm. in diameter and behind that a smaller one.

TABLE 1.—*Tuning Fork Tests (Case 1)* *

Vibrations	Normal	Before Treatment		Ten Minutes after Treatment with Heat Bougie
		Right	Left	Left
64	40	30	35	35
	100	15	25	60
128	35	30	23	30
	60	30	38	48
256	20	15	12	11
	30	15	12	14
512	30	15	25	23
	60	48	52	60
1,024	30	28	27	35
	65	60	55	75
2,048	..	..	..	..
	40	33	33	45

* Tuning fork tests are carried out by one person, in a quiet room, as follows: a percussion rod falls from a horizontal position to strike the fork held at a marked line. The fork is applied with approximately the same pressure to the center of the mastoid process, and to a point about 1 cm. lateral to the exterior auditory meatus. The readings are for seconds of time of hearing and are fairly accurate.

One allows a difference of plus or minus five seconds in the interpretation of the records. In the tables the upper figure stands for bone conduction and the lower figure for air conduction. The normal offered here was obtained by averaging the lengths of hearing time of a group of persons with apparently normal hearing tested under the conditions indicated.

The right tonsil was atrophic; the anterior pillar was congested. The left tonsil was also atrophic; the pillar was slightly congested. The pharyngeal mucosa was smooth.

The orifices of both eustachian tubes were open. There was a moderate amount of adenoid tissue. On bougienage one felt a slight stricture at 20 mm. and a more marked one at 28 mm. up from the orifice of the right eustachian tube.

Hearing tests were made on the morning of the examination, one test before treatment with the heat bougie and one ten minutes after treatment.

Treatment and Course.—After use of the heat bougie the patient was instructed to employ routine nasal treatment for one week. At the end of that time tuning fork tests were made again. As the second tests showed no further improvement, it was decided to remove the polyps present.

Since this operation the patient has stated that tinnitus has decreased.

CASE 3.—*History.*—J. S., a school boy aged 17, had had difficulty in hearing, with "clicking" in the ears, for three months; the condition was worse on the right side. He had had no difficulty before this except for a discharge from both ears, which had appeared on and off for a day or two during the previous six months. Head colds were uncommon, there was no difficulty in breathing and the patient had not suffered from headache or sore throat.

TABLE 2.—*Tuning Fork Tests (Case 2)*

Treatment	Normal	Before Treatment		After Treatment, Right	One Week Later	
		Right	Left		Before Treatment, Right	After Treatment, Right
64	40	10	10	28	25	25
	100	40	60	70	70	63
128	35	20	25	23	20	23
	60	40	55	60	55	55
256	20	8	8	3	9	8
	30	13	13	13	15	10
512	30	18	15	28	20	23
	60	50	50	55	62	62
1,024	30	25	23	33	35	33
	65	60	75	62	65	65
2,048	..	..	..	..	..	..
	40	25	35	25	25	22

TABLE 3.—*Tuning Fork Tests (Case 3)*

Vibrations	Normal	Before Treatment		After Treatment, Right	One Week Later		
		Right	Left		Before Treatment		After Treatment, Left
					Right	Left	
64	40	35	40	45	35	60	55
	100	35	85	70	65	15	110
128	35	40	35	45	45	40	40
	60	45	45	60	60	15	45
256	20	12	12	20	15	10	15
	30	15	14	25	15	12	20
512	30	40	30	35	35	35	40
	60	55	50	60	55	50	60
1,024	30	45	30	40	45	38	40
	65	45	60	65	63	70	80
2,048	..	..	..	..	..	..	..
	40	35	45	35	40	40	45

Examination.—The right ear drum was thickened and scarred; the landmarks could not be recognized. The superior portion of the drum was slightly reddened. There was a questionable discharge inferiorly and posteriorly. The drum was fixed. The left drum was thickened and scarred; the landmarks were not recognized. There were calcium plaques on the posterior portion. The drum was well fixed.

The nasal septum was in the midline, with some deflection posteriorly to the right and with a spur anteriorly. The tuberculum was thickened on the left. The right inferior turbinate was very large, of good color and slightly edematous; the posterior end was markedly hyperplastic. The right middle turbinate was greatly

enlarged; the anterior portion was congested, while the posterior three fourths was highly hyperplastic. The left inferior turbinate was large and normally pink; the posterior portion was hyperplastic. The left middle turbinate was of medium size; it was slightly congested anteriorly, and there was marked hyperplasia in the posterior fourth. The orifice of the right eustachian tube was open; that of the left was closed by edema of the surrounding tissue.

The right tonsil was flat, small and cryptic; the anterior pillar was slightly congested. The left tonsil was like the right one. The posterior pharyngeal wall was atrophic, dry and slightly congested.

Treatment and Course.—At the patient's first visit the right ear was treated with the heat bougie and immediately thereafter by inflation. The inflation forced out a small amount of gelatinous white discharge.

At the second visit the left ear was treated with the heat bougie only.

Hearing in the left ear was practically normal; that in the right ear was slightly below normal, and remained so. The patient had routine nasal treatment for a while previous to treatment with the heat bougie, but his hearing was not improved.

TABLE 4.—*Tuning Fork Test (Case 4)*

				One Week Later		
		Before Treatment		After Treatment with Heat Bougie to Left Ear		After Treatment with Heat Bougie to Right Ear
Vibrations	Normal	Right	Left	Right	Left	
64	40	25	35	30	40	30
	100	70	70	105	110	95
128	35	35	30	25	30	40
	60	60	45	60	65	65
256	20	10	14	10	13	13
	30	15	20	12	15	15
512	30	35	35	25	20	25
	60	65	55	65	60	60
1,024	30	45	40	45	40	45
	65	70	68	76	72	90
2,048	..	..	..	..	..	..
	40	35	40	33	33	45

CASE 4.—*History.*—Miss H. Y., a secretary aged 44, suffered from fulness in the ears and loss of hearing, which were worse on the right side. After a swimming episode, which occurred one year before I first saw her, she had had boils in her right ear. Both ears had "discharged" on and off, twenty to thirty times, during that year. She had had frequent and regular treatments to the ears, particularly with ultraviolet rays. She had had headache on and off for fifteen years, worse in the right occiput, the frontal region and the crown, less on the left side. Nose colds had been infrequent; the patient had had no difficulty in breathing since having an operation on the nose, which had also relieved the pain in the frontal region.

Examination.—The right ear drum was thickened and desquamated; the landmarks were gone. The drum was also retracted. The external auditory meatus showed thickened, fissured and dry skin throughout. The left ear drum was fairly pale and retracted. It was well fixed, especially anteriorly.

The nasal septum was in the midline; the tuberculum was thickened on the right side. A submucous resection had been done. The right inferior turbinate was large and pale. The right middle turbinate was congested and boggy. The

left inferior turbinate was small and light pink; the posterior end was markedly hyperplastic. The left middle turbinate was very large, was fairly edematous and had crusts on it. The orifices of both eustachian tubes were open. A moderate amount of adenoid tissue was present. The hearing test made at the time of examination showed essentially a conductive type of deafness, though there was a slight drop in hearing for the 2048 note.

Treatment and Course.—Routine nasal treatment was prescribed. At the end of a week the heat bougie was applied to the left ear, and at the end of the following week to the right ear.

This was a case of conductive deafness, in which the condition was not improved by nasal treatment. Immediately after use of the heat bougie to the left ear there was better hearing in that ear. The next week the right ear was treated with the bougie. There was much less fulness. Eczema of the auditory canal cleared without treatment; it was probably due to sea food allergy.

Photograph of the heat bougie in position for treatment of a patient. *A* indicates the galvanometer of the thermocouple, *B*, the rheostat for regulating the heat and *C*, the heat bougie proper.

COMMENT

The danger in using the heat bougie is negligible. In my first report I mentioned 2 cases in which a stricture was produced in the tube by overheating but the passage was reopened completely by the heat bougie. Since those incidents, which occurred early in the use of the bougie, there have been no mishaps in over eight hundred treatments. One caution is necessary: Do not insert the bougie into the tube over 35 mm.; otherwise the middle ear is heated and the semicircular canals are quickly stimulated. The first heat bougie used did not have a thermocouple at the distal end to record the temperature of the heated mucosa. The heat was regulated by turning the dial of the rheostat to any point between 110 and 120 F.

For the past five years I have been using the heat bougie with the thermocouple in the distal portion. Lately I have felt that the original,

simple form of the bougie, that is, without the thermocouple, is as effective. However, instead of raising the temperature to 110 to 120 F. one must raise it to 130 to 140 F., which is the degree of heat these fine bougies require to raise the temperature in the mucosa of the tube to 105 to 110 F.

There is a group of otologists who think that treatment of the eustachian tube is useless. Shambaugh,[4] in a study of over 5,000 severely deaf children, wrote:

> It is of interest to note that the serious defects in hearing resulting from the infectious fevers, such as scarlet fever and measles, are not dependent to any extent on the occurrence of otitis media; rather, they are the result of toxic neuritis which develops independent of any infection of the middle ear.

Similarly, in a study of 165 cases of severely deaf adults, Shambaugh and his associates[5] said that deafness in 70 per cent of the cases was due to otosclerosis, in 18 per cent to neuritis of the cochlear nerve and in the few remaining cases to infections of the middle ear.

The percentages of otosclerosis and of cochlear neuritis that Shambaugh and his associates recorded are unusually high; no one else has reported such high figures. They used the term progressive deafness interchangeably with otosclerosis and placed the patients with progressive deafness in the otosclerotic group unless it was proved that they belonged elsewhere. One wonders whether many of these cases of progressive loss of hearing are not cases in which deafness from gradually progressing chronic otitis media develops to a severe degree because of fixation of the ossicular chain, particularly of the stapes.

Many, perhaps most, otolaryngologists thinks that otitis media is intimately associated with, if not dependent on, infection of the nasal and the sinus mucosa. Hoople and Cave,[6] in a study of two series of cases of scarlet fever in children and in adults in a pavilion for patients with a contagious disease, pointed out:

> There were 33 cases with otitis media among the 292 examined. . . . This table [table 5] brings out the interesting fact that no ear showed purulent otitis media unless there was a sinusitis evident by the x-rays on the corresponding side.

Of the second group they wrote:

> Twelve cases, or 15 per cent of the group [80 cases], were complicated by otitis media. It will be seen from table 10 that there was no involvement of the ear without involvement of the sinus, and in every case but one, the involvement was on the same side as the otitic lesion.

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4. Shambaugh, G. E.: Disease of Ear in Childhood, Am. J. Dis. Child. **39**: 1062 (May) 1930.

5. Shambaugh, G. E.; Wallner, L. J.; Greene, L. D., and Shambaugh, G. E., Jr.: A Clinical Study of Severe Deafness in Adults, Arch. Otolaryng. **18**:430 (Oct.) 1933.

6. Hoople, D. G., and Cave, L. S.: Otorhinologic Aspect of Scarlet Fever, J. A. M. A. **101**:1121 (Oct. 7) 1933.

Campbell [7] offered a somewhat similar conclusion in his observations on a group of infants and children:

> A report of a rather small number of cases [150] in which acute sinusitis was found in practically 100 per cent of the cases of acute otitis media does not necessarily prove that all cases of otitis [media] are secondary to sinusitis, but it at least suggests the common association of the two conditions. Furthermore, it suggests that in cases of otitis media the treatment should not be confined to the ear, but should be largely directed to the elimination of the sinus infection.

Fowler,[8] in a study of 100 cases, including cases of chronic otitis media of all varieties, chronic suppurative, chronic catarrhal and healed and recurrent suppurative, and of nerve deafness, found that "eighty-six per cent showed positive pathologic changes in the sinuses."

Many otolaryngologists think of chronic sinusitis as a somewhat uncommon, at least a not ubiquitous, inflammation. Others have attempted to indicate the prevalence of this ailment, particularly the chronic type. Hoople and Cave [6] stated with reference to their first group of cases of scarlet fever, in which they took roentgenograms sometime after the patients were admitted to the hospital: "Ninety-one per cent of the total [292 patients] showed some shadow on the x-ray film; only 9 per cent showed all the sinuses clear." (Eighty-three and nine-tenths per cent showed thickened membrane; 7.2 per cent retained secretions.) Realizing the high frequency of sinusitis in these cases, they took roentgenograms in the second group both on admission of the patients to the hospital and on their discharge. They recorded that on admission 91 per cent of the 80 patients had roentgen evidence of sinusitis; at the end of the period of isolation 58 per cent of these patients had some involvement of the sinuses.

Kern and Schenck,[9] in a study on the evaluation of roentgenographic and clinical examination in the diagnosis of sinusitis, stated that they had been struck by the prevalence of sinsusitis in so-called normal persons. They followed up this work, and in a second report purported to show the ubiquity of sinusitis. They stated:

> Several years ago we [10] studied . . . a group of fifty persons (table 1). They gave no history of respiratory disease occurring within six months, of susceptibility

7. Campbell, E. H.: Association of Acute Sinusitis and Acute Otitis Media in Infants and in Children, Arch. Otolaryng. **16**:829 (Dec.) 1932.

8. Fowler, E. P.: Incidence of Nasal Sinusitis with Disease of Ear, Arch. Otolaryng. **9**:159 (Feb.) 1929.

9. Kern, R. A., and Schenck, H. P.: Chronic Paranasal Sinus Infection, Arch. Otolaryng. **18**:425 (Oct.) 1933.

10. Kern, R. A., and Schenck, H. P.: The Relative Efficiency of the Clinical and the Roentgenologic Methods for Sinus Disease Diagnosis, Am. J. M. Sc. **178**: 168 (Aug.) 1929.

to colds or of frank sinus disease, and their ages fell proportionately into the same decades as those of our first two hundred asthmatic patients. A group so selected may well be considered as better than average. Yet 72 per cent gave roentgen evidence of abnormality of the sinuses, and in 26 per cent the abnormality was clinically detectable. There is every reason to believe that these abnormal findings may be evidence of sinus disease in the past rather than of active sinusitis. But it seems to be clear that three fourths of the population have or have had at some time in their lives a protracted or a severe sinus infection.

Even from my limited observations I feel that chronic sinusitis is a common ailment. Hyperplastic changes in the mucosa of the nose and of the nasal sinuses are present in the majority of adults. In some persons careful search is required to locate areas of hyperplasia, but a routine nasopharyngoscopic examination, particularly of the middle turbinate, the meatus and the posterior portion of the inferior turbinate, will show how frequently these pathologic changes are encountered. perhaps otologists have too fixed an idea that pus must be present if a diagnosis of sinusitis is to be made.

The frequency of occurrence of chronic otitis media (nondischarging) is greatly underestimated by most otologists. Few, probably, would ever think that a person with normal hearing and apparently normal sinuses had any pathologic condition in the middle ear. One easily assumes that only a patient with loss of hearing may be considered to have chronic (nondischarging) otitis media. Polvogt,[11] after a careful study of microscopic sections of middle and inner ears, pointed out differently:

As the result of this study of sixty-three ears from patients who had a test of hearing shortly before death and whose hearing was found to be normal, it is certain that marked pathologic changes of the tympanic membrane may exist without causing any noticeable impairment of hearing.

In this same group 80 per cent of the ears showed strands of embryonic tissue in the region of the oval and of the round window; the strands were usually more fibrous in the region of the round window and in some instances were attached to the secondary tympanic membrane. Polvogt also noted that "an abnormal degree of thickening of the mucous membrane over the region of the promonitory was present in sixteen [25 per cent] of the sixty-three ears." Polvogt and Bordley,[3] in a second study based on examination of sections of middle and inner ears from 40 persons whose hearing had been tested within several months of their death and who had had either normal hearing or a conductive deafness, reported: The ears of all the 20 persons who had given a negative reaction to the Rinne test had lesions in the fossula of the oval window. Seventeen of 20 apparently normal ears had

11. Polvogt, L. M.: Histologic Variation in the Middle and Inner Ear of Patients with Normal Hearing, Arch. Otolaryng. **23**:48 (Jan.) 1936.

lesions in this same area, but not to such a great extent as those ears with conductive deafness. Of this same group of normal ears the pars flaccida in 11 of 20 and the pars tensa in 10 of 20 were involved, but the lesions were not as great as those found in the fossula of the round window. It is evident that these pathologic conditions are much more prevalent than otolaryngologists usually believe. A lesion may be trivial and still produce marked loss of hearing; on the other hand, one might have scarring and fibrosis of fairly marked degree and still have normal hearing. The first view has been expressed by Fowler,[12] reporting on a detailed examination of a large number of temporal bones, some from his own collection and some borrowed from Johns Hopkins University School of Medicine. Some of the specimens showed otosclerosis, while others showed indications of chronic otitis media. Fowler said:

> These all show some dense fibrous tissue or calcified connective tissue, suggestive of actual or past masked inflammation. This means that middle ear disease is an extremely common disease, much more common than is generally supposed.

When one considers treating any of these conditions with the heat bougie, naturally one is extremely skeptical of overcoming scarring and fibrous adhesions of the ossicles and windows by opening the eustachian tube or in fact by any form of therapy known at present.

Direct treatment to the eustachian tube is not effective in all cases. As has been stated, the efficacy of opening the tube depends on whether stapedial fixation and fibrosis around the ossicular chain are present. There are many cases in which, fortunately, the stapes and other parts of the ossicular chain move freely and in which hearing is improved by treatment of the eustachian tube. This condition can be ascertained only by opening the tube. Relief from tinnitus depends on the cause of the condition; the type of tinnitus that is aided by opening of the tube, that is, the type in which aeration of the middle ear will give relief, is fortunately a fairly common type. I feel, as many others do, that frequently much can be done by relieving or improving inflammation in the nose, nasopharynx and sinuses, which is a common chronic ailment. After comparison of the methods used in direct treatment of the eustachian tube, namely politzerization, inflation by means of a catheter, the Valsalva method and use of a regular bougie, a Yankauer applicator, an electrolytic bougie or the heat bougie, I feel that use of the heat bougie will open the tube in every case and keep it open for a longer time than any of the other procedures. It is the most effective direct method for treatment of catarrhal deafness. However, it certainly is not a cure-all.

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12. Fowler, E. P.: Otosclerosis with Other Lesions, Tr. Am. Otol. Soc. **23**: 143, 1933.

CONCLUSIONS

1. Use of the heat bougie will open the eustachian tube for longer intervals than any other procedure. Opening up the eustachian tube helps only those patients who have no scar tissue in the middle ear restricting movement of the ossicular chain.

2. Chronic hyperplastic sinusitis is an extremely prevalent disease. Chronic nonpurulent otitis media is very common and is probably dependent on chronic nasal infection or chronic sinus infection (with its occasional acute flare-ups) for its source. These inflammatory processes are present in varying degree in most adults; they may exhibit no symptoms, slight symptoms or marked symptoms.

3. Chronic so-called catarrhal deafness may be helped by improving the rhinosinal mucosa and by direct treatment of the eustacian tube. Unfortunately, there is at present no known specific way to cure chronic sinusitis. One can, by various empiric methods, improve the condition of the mucosa in the nose and in the sinus to some degree, in most cases for a limited time, and thereby alleviate or temporarily stop the inflammatory process in the ear.

4. Direct treatment of the eustachian tube, particularly with the heat bougie, is of value in many cases of chronic catarrhal otitis media, but not in all, since it does not remove ossicular restriction. Aeration of the middle ear does help some patients, even if only temporarily; it seems to tide others over acute tubotympanic catarrh, leaving the ears apparently well thereafter. In some cases the condition gets worse regardless of all treatments. This is because one is unable to halt the course of hyperplastic sinusitis which is the source and cause of chronic progressive otitis media.

238 Lark Street.

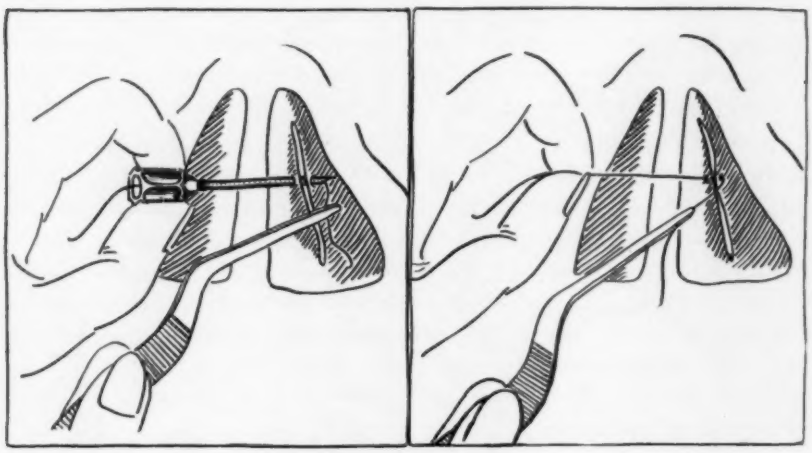
Clinical Notes

SIMPLIFIED INTRANASAL SUTURE

J. H. Childrey, M.D., San Francisco

The following method of suturing within small cavities is valuable in intranasal operations, particularly in submucous resection. It is time saving, does away with the need for an assistant and overcomes the disadvantage of the scant space in the nostril.

Suturing the flaps of nasal mucosa is difficult when the ordinary curved or partly curved needle and the needle holder are used, because the nostril is too small to permit free manipulation of the instruments. The material (dermal suture is preferred because it is rigid enough to be threaded easily) is threaded



Intranasal suture.

through the lumen of a no. 20 hypodermic needle 1½ inches (3.8 cm.) long. The needle, held in the fingers, is directed away from the operator; the point is inserted first through the margin of the proximal flap, and then through the margin of the distal flap. When the distal end of the suture appears it is caught and held with a bayonet or other forceps and the needle is withdrawn backward. The suture, held with the forceps, is drawn farther through, and the knot is tied.

Greens' Eye Hospital.

PRECISION AND SAFETY IN INTRANASAL COCAINE ANESTHESIA

Max Unger, M.D., New York

I began the practice of otolaryngologic surgery in the late Dr. Emil Mayer's clinic over twenty-five years ago and had the benefit of Dr. Mayer's careful and thorough instruction in his specialty. I also had close hand knowledge of the work

on the toxic effects of local anesthetics done by Dr. Mayer and his associates[1] for the Council on Pharmacy and Chemistry of the American Medical Association. With the precepts and recommendations of Dr. Mayer and his committee in mind, I did many intranasal operations with cocaine anesthesia without a mishap and almost reached the state of mind where I thought "This can't happen here." Well, it did happen. During the course of a submucous resection of the nasal septum, symptoms of acute cocaine poisoning developed. The patient recovered after desperate and intensive work, but the experience was so harrowing that I was impelled to make another study of Dr. Mayer's report and of my own experience and observation, in the hope that something might be discovered or devised to safeguard further the use of cocaine as an anesthetic in the nose. I am happy to state that I have developed a procedure that involves precision in the application of cocaine in the nose, reduces the amount required for adequate anesthesia and, therefore, adds to the safety of the patient.

A glance at drawings *A* and *B* in figure 1 will provide a graphic idea of the anatomic basis of my method. These drawings show diagrammatically the nerve supply of the septum and the lateral wall of the nasal cavity. A line drawn from the anterior-superior border of the sphenoid bone to the anterior nasal spine divides, roughly, the septum and the lateral nasal wall into halves, an anterior-superior and a posterior-inferior half. The nerve supply of each half enters the nose at a different point. The anterior-superior half of the septum is supplied chiefly by the internal branch of the nasal nerve (a branch of the ophthalmic nerve). The anterior-superior half of the lateral wall of the nose is supplied by the external branch of the nasal nerve. These two nerves enter the nasal cavity through a slit in the cribriform plate of the ethmoid bone, at the top of the vault of the nose, just back of the nasal process of the frontal bone. The vault of the nose at this point, as seen in drawing *C*, is between 1/16 and 1/8 inch (0.16 and 0.32 cm.) wide, and a pledget of cotton soaked with a solution of cocaine hydrochloride and applied at this point will anesthetize both nerves.

The posterior-inferior half of the septum is supplied by the nasopalatine branch of the sphenopalatine ganglion, which enters the nasal cavity by the sphenopalatine foramen and runs across the lower anterior border of the sphenoid bone to the septum and diagonally downward and forward to the anterior palatine canal. The posterior-inferior half of the lateral wall of the nose is supplied by the superior nasal branches of the sphenopalatine ganglion and by the branches from the anterior palatine nerve. All these nerves enter the nasal cavity through the sphenopalatine foramen, located at the lower anterior border of the sphenoid bone where it joins the lateral wall. The width of the nose at this point, as shown in drawing *D*, is between 1/4 and 3/8 inch (0.64 and 0.96 cm.), and a pledget of cotton soaked in solution of cocaine hydrochloride and applied to the lower and inferior border of the sphenoid bone will anesthetize the posterior-inferior half of the septum and the lateral wall of the nose.

The technic used is as follows:

A piece of absorbent cotton is rolled loosely into a cylinder about 3/4 inch (1.91 cm.) long and 1/4 inch (0.64 cm.) thick and tied in the middle with a thread, one or both ends of the thread being left about 5 inches (12.5 cm.) long. A 4 per cent solution of cocaine hydrochloride is drawn into a measuring pipet or into a syringe with millimeter or minim readings (a tuberculin syringe does nicely). Three cubic millimeters exactly (0.3 cc.) of this 4 per cent solution is dropped on

1. Mayer, E.: Toxic Effects Following the Use of Local Anesthetics, J. A. M. A. **82**:876 (March 15) 1924.

the cotton roll. The ordinary grade of cotton absorbs this completely, so that the fingers are barely dampened by handling it. If anesthesia is wanted in the anterior-superior half of the nose, the cotton roll is pushed up to the front part of the vault of the nose and left there, with the cotton thread hanging through the nostril. If anesthesia is desired in the posterior-inferior half of the nose, the cotton roll is

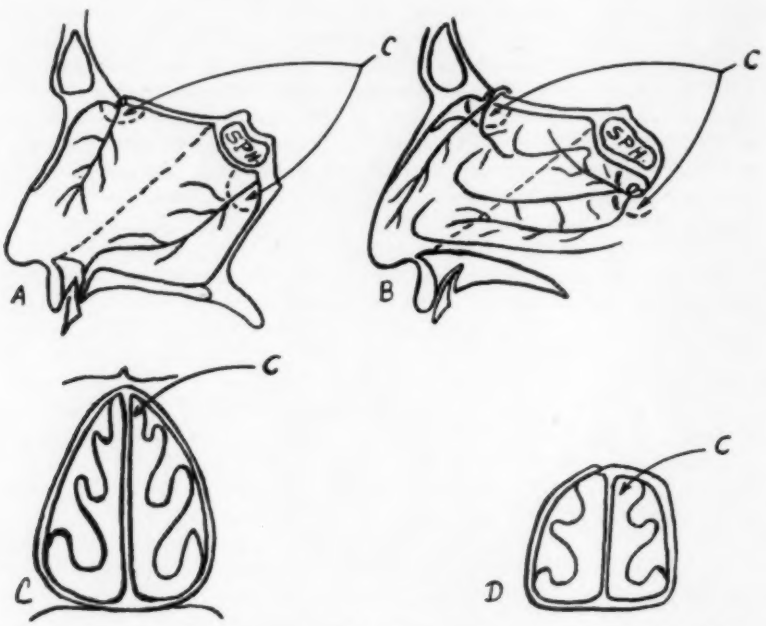


Fig. 1—*A* shows the nerve supply of the septum and *B* that of the lateral wall; *SPH.* indicates the sphenoid bone. *C* shows the vault of the nose at the front and *D* the vault under the sphenoid bone. The lines indicated by *c* in the four drawings point to the situation of the cocainized cotton roll.

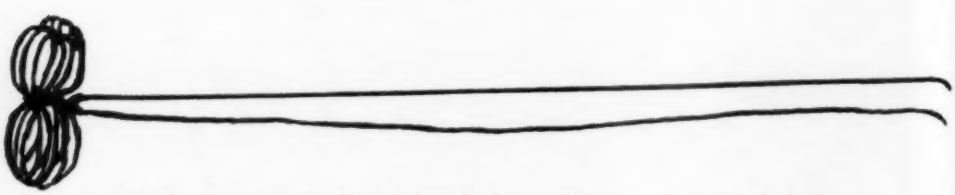


Fig. 2.—The cotton roll and threads for applying cocaine; actual size.

pressed against the inferior border of the sphenoid bone. Anesthesia is complete in fifteen minutes and is felt by the patient as numbness, first in the nose and then in the gums and the tip of the nose.

The exact size of the cotton roll is shown in figure 2. The threads are left hanging through the nostrils to facilitate removal.

One cotton roll with 3 cu. mm. (4.5 minims) of 4 per cent solution of cocaine hydrochloride will contain 0.012 Gm. (about ⅕ grain) of cocaine. If four cotton

rolls are used to anesthetize both sides of the nose, as for a resection of the septum, the total solution put in will be 12 cu. mm., containing a total of 0.048 Gm. of cocaine (about ¾ grain). Dr. Mayer's committee gave 1½ grains (0.096 Gm.) as the outside limit, so that the quantities mentioned are well within the safety limit.

I do not add epinephrine, because I feel that it detracts from the anesthetic qualities of the cocaine and I distrust its synergistic action. Besides, cocaine itself is a pretty good vasoconstrictor and does not cause so much after-congestion. The blood vessels supplying the nose enter it at the same place as the nerves and will be constricted by the same pledget of cotton.

I made a cursory search of the literature for priority in the use of this method without finding any mention of it. Priority is really not important. Even if a dozen physicians anticipated me in using this procedure, it is still a good method.

226 West Forty-Second Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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PLASTIC SURGERY, 1936-1938

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AND
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A review of the progress made in plastic surgery during 1936 and 1937 encompasses many phases of activity. The impetus of an original discovery greatly accelerates and often revolutionizes a whole field of endeavor. In the interim, modification and internal development continue to test what is sound and worthy of survival. As plastic surgery touches in interest several fields, such as general, ophthalmic, maxillofacial and otorhinologic surgery, the interrelationships favor an interchange of ideas and methods. Not only do the frontiers of a specialty change with time and development, but the internal flux tends to organize the members within the group to evolve standards of practice and of the educational fitness of new members. This paper is an attempt to cover these various phases of activity in plastic surgery in the light of the recent literature.

SCOPE AND PROGNOSIS IN PLASTIC SURGERY

Gillies and Mowlem,[1] in a paper on the scope and the prognosis possible in plastic surgery make the following pertinent remarks:

> The great majority of congenital lesions or defects can be eliminated or materially improved. In most instances treatment should begin in early life. This will assure the best possible result from the physical standpoint, and will reduce to a minimum secondary psychological traumata. . . . Acquired defects may be the result of trauma, of disease, or the treatment of disease. In this latter category come defects created in the removal of disease by surgical means and also those which sometimes follow the use of radium and X rays and such cauterising agencies as diathermy and carbon dioxide snow. Trauma, however, is the commonest cause, and the amount of damage inflicted necessarily varies from minor abrasions to extensive loss of tissues, which if untreated will cause severe secondary defect.
>
> Immediate suture after débridement naturally offers the best prospect of a good scar. . . . When a wound has healed and has produced an unsightly

1. Gillies, H. D., and Mowlem, R.: Prognosis in Plastic Surgery, Lancet **2**:1346 (Dec. 5); 1411 (Dec. 12) 1936.

scar, improvement can frequently be obtained by excision of the scar and accurate resuture. . . . Extensive lacerations should be treated as emergencies. If accurate débridement is carried out, if all tissue of doubtful viability is removed, and if the resultant raw area is immediately provided with a new skin covering, then healing by first intention should be the rule. . . . Healed tissue losses result in cicatricial contractures. On the face this may cause ectropion of lids or lips, or gross distortion of other structures. . . . As a rule such a scar must be removed in its entirety, and all normal parts allowed to resume their correct position. The resultant defect is then made good either by some form of free graft or by a pedicle flap brought in from a distant part. . . .

Fractures of the mandible or of the maxilla should be dealt with as early as possible. There is no necessity to wait for the subsidence of swelling, since in most cases the occlusion of the teeth will afford the most accurate guide as to the accuracy of the reduction. . . .

The loss of nose, lips, ears, or other specialised structures, whether as the result of accident or disease, can usually be made good. As a rule these repairs must necessarily be carried out at the expense of some other part of the patient's anatomy.

Beck [2] traces the development of plastic surgery and outlines its aim as a truly scientific and honest specialty, practiced by preeminently skilful, qualified and honorable members of the medical profession. He speculates on the undreamed of possibilities which will open up for physiologic reconstructive surgery when the problem of tissue compatibility is solved.

In discussing the need for well trained "contour surgeons," Blair [3] expresses the opinion that their basic training should be a thorough grounding in general surgery.

In an examination of the interrelationships of the plastic surgeon and the otolaryngologist, Hays [4] summarizes the plastic procedures which he feels should remain the intimate problem of the otolaryngolosist, i.e., repair of tracheotomy wounds, freeing of adhesions of the posterior pillar of the fauces to the pharyngeal wall, removal of tonsils and adenoids in patients with cleft palate, closure of perforations of the nasal septum, plastic operation for either congenital or acquired atresia of the auditory canal, correction of prominent ears and closure of deep clefts following operations on the mastoid.

GRAFTING METHODS

Dufourmentel [5] reviews the physical and physiologic requirement of successful skin grafting. He particularly stresses the importance of

2. Beck, C.: Aims and Future of Plastic Surgery, Am. J. Surg. **31**:1 (Jan.) 1936.

3. Blair, V. P.: Specialty Surgery and "Plastic Surgery," Surg., Gynec. & Obst. **62**:895 (May) 1936.

4. Hays, H.: Relationship Between the Otolaryngologist and the Plastic Surgeon, Am. J. Surg. **31**:38 (Jan.) 1936.

5. Dufourmentel, L.: La destinée des greffes libres, Rev. de chir. structive, December 1937, p. 371.

meticulous attention to the condition of the field over which the graft is to be laid.

Grafting of skin from one person to another has always been an ideal in plastic surgery. Some patients with marked deformity do not have sufficient healthy or conveniently available tissues for grafting. Others have been so debilitated by the original injury and attendant infections that various grafting procedures impose a considerable surgical burden.

Moran [6] has recently undertaken to transfer a tubed graft raised on a normal healthy Negro boy to his badly burned cousin, who has the same type of blood. Gillies [7] has tried three similar operations, all of which have been failures. In one case a biopsy specimen was taken from the skin on each side of the normal-looking joining scar, a portion of the scar being included. Section showed blood vessels going up to the scar but failing to penetrate the donor skin. Gillies suggests that the success of cartilage and cornea transplants is due to the fact that neither of these substances depends on the presence of blood vessels for nutriment and that tissues of the body which appear to require blood vessels cannot as yet be grafted from one patient to another.

Similarly, the transfer of free skin grafts from one person to another has, with possible exceptions, been a failure. Ashley,[8] however, reports the successful use of foreskins as skin grafts. With a standard method of grafting, he used foreskins obtained from infants circumsized when they were from 7 to 10 days old. The grafts were preserved for several days in physiologic solution of sodium chloride in a refrigerator or embedded in ice cubes. No cross agglutination to determine blood types was done. Nineteen of a total of twenty-seven foreskins in 1 case and two of four in another case grew well and fused with one another and with the edge of the denuded areas.

Some interesting observations on the fate of deeply embedded dermal grafts were made by Peer and Paddock.[9] The sebaceous glands and the hair follicles disappeared early. The sweat glands ultimately underwent degeneration and fibrous replacement.

The transplantation of cartilage from the maternal ear to the deformed ear of the offspring has been successfully carried out by

6. Moran, R. E.: Artificial Siamese, Time **30**:30 (Dec. 13) 1937.

7. Gillies, H. D.: Reconstruction of the External Ear with Special Reference to the Use of Maternal Ear Cartilage as the Supporting Structure, Rev. de chir. structive, October 1937, p. 169.

8. Ashley, F.: Foreskins as Skin Grafts, Ann. Surg. **106**:252 (Aug.) 1937.

9. Peer, L. A., and Paddock, R.: Histologic Studies of the Fate of Deeply Imbedded Dermal Grafts: Observations on Sections of Implants Buried from One Week to One Year, Arch. Surg. **34**:268 (Feb.) 1937.

Nattinger [10] and by Gillies.[7] Homologous and autologous cartilage and, in some instances, such heterologous substances as ivory have been used successfully in plastic operations on the nose. The majority of operators favor autologous cartilage as best fulfilling the esthetic and the physiologic requirements of such operations, but the ease of using prepared grafts of heterologous cartilage and the fact that in the number of "takes" this material compares favorably with fresh autogenous material have won for its use such advocates as Spanier [11] and Straith and de Kline.[12]

Transplantation of osseous tissue from one part of the body to another part is usually successful. The work of Orell [13] in transplanting homologous and heterologous osseous tissue has created a new terminology and new hope in this field. His "os purum" is defined as bone freed from fat, connective tissue and proteins by means of lengthy physiochemical processes, but it is not freed entirely from all the cartilaginous matrix. "Os novum," in contradistinction to "os purum," is composed of the calcium framework of dead bone. It is produced by implanting a narrow splint of "os purum" over the anteromedial surface of the tibia and excising it from one to two months later. Boiled fresh bone differs from "os purum" in that it contains fat, connective tissue and proteins.

These materials were used in cases of tuberculosis of the osseous system and in cases of osteomyelitis of parts other than the face or jaws, and at present, while there is evidence of their distinct value, a proper evaluation cannot be drawn. This development if successful may well find application in maxillofacial work.

Several modifications and improvements in the technic of skin grafting are suggested. Coelst [14] has substituted a transparent celluloid frame on which to place the graft for the usual modeling compound. Day by day inspection of the graft is possible with this method.

A method of holding skin grafts firmly on the graft bed is described by Trueblood,[15] who uses a dressing of paraffin which has been sprayed

10. Nattinger, J. K.: Total Reconstruction of the External Ear, Northwest Med. **36**:172 (May) 1937.

11. Spanier, F.: Die Verwendung von allo-und heteroplastischen Material bei der Wiederherstellung von partiellen Gesichtsdefekten besonders bei Sattelnasen, Rev. de chir. structive, December 1936, p. 391.

12. Straith, C. L., and De Kline, E. H.: Modern Management of the Fractured Nose, Internat. Abstr. Surg. **66**:9 (Jan.) 1938.

13. Orell, S.: Surgical Bone Grafting with "Os Purum," "Os Novum" and "Boiled Bone," J. Bone & Joint Surg. **19**:873 (Oct.) 1937.

14. Coelst, M.: Une autre application de ma méthode de "outlay sur cadre," Rev. de chir. structive, March 1936, p. 195.

15. Trueblood, D. V.: Paraffin Dressing for Transplanted Grafts, West. J. Surg. **44**:578 (Oct.) 1936.

on and allowed to solidify, and also by Taylor,[16] who utilizes a football bladder with a stand-pipe which can be filled with water and the pressure regulated to the optimal 15 cm.

The solution of various mechanical problems met with in grafting, such as supports for donor areas, an apparatus for carrying grafts and scar-softening appliances, were ingeniously devised by Darcissac.[17]

A method of securing small grafts with the minimum of donor deformity is described by Zee,[18] who removes strips of skin about 4 by 10 cm. in size, undermines the edges and closes the donor wound by primary closure. Such a strip when cut up into small pieces will yield about thirty grafts, the number necessary to cover about 25 sq. cm.

Conway[19] removes his small grafts from an elliptical area to the point of complete denudation of the area, so that it is possible to undermine and close the wound entirely and have an excellent cosmetic result.

Sammis[20] has modified the removal of full thickness grafts by leaving one, two or more narrow parallel strips of skin on the donor area to act as islands, establishing centers from which healing begins to take place very easily postoperatively.

With regard to tubed flaps, Davis and Kitlowski[21] have changed the initial lateral incisions, so that one ends at a higher level than the other, to allow a perfect closure of all raw areas.

PLASTIC OPERATIONS ON THE FACE

Blair, Brown and Byars[22] stated that "excepting trauma of vital organs and their essential coverings, the final outcome of no injury is so directly dependent upon early proper care as injury of the face." With regard to the soft tissues of the face, they suggest that the important things are not to sacrifice any tissue at the time of injury and not to place and retain sutures that leave scars. Trap door flaps with beveled

16. Taylor, F.: Pressure Bag in Skin Grafting, Am. J. Surg. **33**:328 (Aug.) 1936.

17. Darcissac, M.: La prosthèse pré-et post-opératoire dans l'application des greffes cutanées, cutanéo-muqueuses, cartilagineuses ou osseuses et dans l'amelioration des cicatrices, Bull. et mém. Soc. d. chirurgiens de Paris **28**:424 (June) 1936.

18. Zee, Z. U.: A New Method for Obtaining Small Deep Grafts, Chinese M. J. **50**:935 (July) 1936.

19. Conway, J. H.: Technical Details in Skin Grafting, Surg., Gynec. & Obst. **63**:369 (Sept.) 1936.

20. Sammis, G. F.: New Method for Transfer of Full Thickness Skin: Grille Graft, Am. J. Surg. **36**:46 (April) 1937.

21. Davis, J. S., and Kitlowski, E. A.: A Method of Tubed Flap Formation, South. M. J. **29**:1169 (Dec.) 1936.

22. Blair, V. P.; Brown, J. B., and Byars, L. T.: Early Local Care of Face Injuries, Surg., Gynec. & Obst. **64**:358 (Feb.) 1937.

edges, if clearcut, should be sutured into place immediately. The mandate for immediate suture also holds for three other types of injuries: (1) cuts into or through the substance of the ear, especially when a large fragment remains attached by only a narrow pedicle; (2) injuries to all narrow double-surfaced flaps, such as the border of the lip, an ear or an eyelid, and (3) all cuts or tears of the nasal skin and cartilages, including the septum. Roy [23] reports a case of complete avulsion of the nasal ala in which the ala was sutured in situ three hours later and the free graft survived completely.

In discussing treatment of the bony part of the face, Blair, Brown and Byars state that for fractures of the lower jaw when teeth are present, interdental wiring is the safest, simplest and most universally accepted form of treatment. This is also the method of choice for treating fractures of the neck, the condyle and the intermuscular part of the ramus. Fractures of edentulous jaws are usually treated by circumferential wiring of the lower jaw to the patient's lower dental plate and attachment of the lower to the upper plate if necessary. In the reduction of fractures of the maxilla with involvement of the alveolar bone, the displaced fragments and the teeth which are still attached should be replaced as accurately as possible and retained either by wires or by some other dental appliance.

For fractures of the malar bone, which occur with or without associated involvement of the maxillary bone, Blair, Brown and Byars favor the Gillies method, which consists of inserting a long, slender bone elevator into an incision in the skin made at the upper end of the temporal ridge, following down the bony wall of the fossa under the temporalis fascia and prying the bone into position. New [24] uses a small hook inserted through an incision over the depressed bone. Cox [25] presents these two methods, the Matas technic and the intraoral method in his discussion of these fractures.

Nasal injuries are treated as emergencies, the parts being replaced and immobilized as early as possible. Particular attention is paid to recent blows on the nose of an infant or a growing child. Blair, Brown and Byars [22] have made it a rule "in every case of a baby or child so injured to explain the supposed possible results of such an injury, and if permitted, to make forceful pressure on the body bridge from within, on the theory that the later hardening and distorted growth of the septal cartilage which seems to be the basic factor in these develop-

23. Roy, J. N.: Lobule nasal sectionné complètement et suturé trois heures après l'accident, Rev. de chir. structive, March 1936, p. 211.

24. New, G. B.: Traumatic Deformities of the Nose and Other Bones of the Face, Surg., Gynec. & Obst. **64**:532 (Feb.) 1937.

25. Cox, G. H.: Treatment of Fractures of the Facial Bones, New York State J. Med. **37**:52 (Jan. 1) 1937.

mental deformities might have been induced by abnormal backward pressure of imperceptibly depressed bone."

There are patients who have suffered severe facial injuries and who, either because of their physical condition or the lack of facilities at the time of the accident or because of a disregard of professional advice, come to the otolaryngologist and the plastic surgeon with old traumatic bony lesions of the face and nose. McIndoe [26] groups such patients according to the type of injury sustained and outlines the treatment appropriate to each type. The most common injury is a fracture deformity of the nose. Two different types are recognized and require different treatment.

Lateral fractures include those which are due to a blow from the side and result in more or less lateral displacement of the nose and the cartilaginous septum but without loss of the bridge line. These are treated by incision in the lateral vestibular walls, elevation of the soft tissues, complete mobilization of the nose by chisel or saw and replacement in the original position. The septum may require coincidental resection for replacement.

The second type includes depressed nasal fractures. It must be admitted that the attempt to reduce old telescoped fractures by refracture and replacement and thereby obtain a normal nasal contour usually fails, although occasionally it may be successful. The deformity is usually best corrected by a graft from the crest of the ilium or from the seventh costal cartilage.

Losses of soft tissues and attendant scar deformities, as well as old traumatic bony defects, often require extensive replacement and correction. Maliniak [27] has studied the problem of facial defects with special reference to the source of skin grafts. The ideal procedure in all cases is to obtain a sliding or rotating graft from neighboring tissues. Whenever possible the dimensions of the flap are reduced to a minimum by serial excision of the deformity. The ascending flap from the cheek provides skin of excellent quality, as does the descending flap from the nasolabial fold and the lateral nasal wall; however, these flaps are subject to the limitations of size. For tubed flaps the forehead has first choice and the skin behind the ear second. A flap from the neck is particularly valuable for replacement of losses in the lower half of the face. A flap from this region can be tubed and the secondary defect easily closed, with the suture line concealed in the natural fold of the neck.

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26. McIndoe, A. H.: Treatment of Old Traumatic Bony Lesions of the Face, Surg., Gynec & Obst. **64**:376 (Feb.) 1937.

27. Maliniak, J. M.: Repair of Facial Defects with Special Reference to the Source of Skin Grafts, Arch. Surg. **34**:897 (May) 1937; Rev. de chir. structive, December 1937, p. 431.

Concerning the merits of various types of free skin grafts, Maliniak favors the thin graft (epidermal and dermoepidermal) for covering a large body surface and full thickness grafts on the face, forehead, eyelids and cheeks and on areas requiring pressure. To cover large surfaces in the neck, a pedicle flap from the chest or abdomen is the most preferable.

Many operations on the face can be done with the area under local anesthesia. Blocking the infraorbital nerve by the extraoral route is presented by Novick and Saghirian.[28] Their technic is to draw a line vertically from the lower eyelid down through the upper lip at a distance of 1 cm. from the inner canthus of the eye and another line from the nasal spine to the outer canthus. The point of intersection of these lines marks the point of entrance of the needle, which is pointed and advanced in a direction upward and backward in relation to the vertical axis of the head and at an angle of 45 degrees to the anteroposterior diameter. (One of us, H. M., tested these directions on 25 cadavers recently and was able to enter the infraorbital canal in all except 2 on the first try.)

Operations on the lip are unsatisfactory or fail, according to Douglas,[29] because of hemorrhage, rough handling of tissues or imperfect adjustment of edges, especially at the vermilion border, and after operation because of the releasing of sutures or infection. To combat these hazards, Douglas has devised a hemostatic suture and holder which will control bleeding and allow good management of the parts.

The fine, almost invisible, suture line has often been the object of more admiration than a well deserving complex restoration. Lack of a broad red scar is often interpreted as an index of the surgeon's care and skill. The development of atraumatic needles, special instruments, fine sutures and particularly a sound physiologic method has been responsible for the advances. Langner's lines are given due respect, and for wounds which run across them, buried sutures to counteract the pull of the fibers are included in the method. Straith[30] has contributed an article on the subcuticular method of suturing, which he uses successfully.

More and more cases of suture of the seventh nerve with successful results are being reported. Accidental palsy of the seventh nerve caused by radical mastoidectomy should be treated by immediate suture. During the first forty-eight to seventy-two hours the nerve will respond

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28. Novick, J. N., and Saghirian, L. M.: Extraoral Method of Regional Anesthesia in the Superior Maxilla, Internat. J. Orthodontia **23**:286 (March) 1937.

29. Douglas, B.: Aids in Surgery of the Lips, Ann. Surg. **106**:293 (Aug.) 1937.

30. Straith, C. L.: Facial Scars, Am. J. Surg. **36**:88 (April) 1937.

to faradic stimulation, so that the ends can be picked up, but later the only way to find the nerve is by dissection; obviously the chances for a good result are reduced with waiting.

The experimental work of Davis and Aries [31] on the prevention of adhesions about repaired nerves and tendons may find an application in operations on the seventh nerve. These workers found that allantoic membrane has the property of lessening the number and severity of the adhesions which form about injured nerves, tendons without sheaths and damaged peritoneal surfaces.

Prognostic experience with Bell's palsy is crystallized by Duel and Tickle,[32] in their statement that 80 per cent of the patients do not lose entirely their faradic response and recover in from four to six weeks, while the 20 per cent in whom the faradic response disappears entirely in from two to three days will not recover and should have an immediate decompression of the seventh nerve.

The treatment of the malignant diseases of the face, nose and mouth has in many cases shifted from the operating room to the roentgen ray laboratory. That the man specializing in cancers of the face should be trained in both surgery and roentgenology is in the best interests of the patient and of the profession. New and Figi [33] recommend that precancerous keratosis and leukoplakia be treated surgically. Malignant processes in the lip which have not involved the neighboring maxilla or mandible and can be excised with a reasonably wide border of normal tissue can be removed and primary suture carried out by means of wedge-shaped full thickness flaps at the corners of the mouth.

Patterson [34] has been dissatisfied with the results of the treatment of carcinoma originating in the mucous membrane of the cheek because of the mutilation and fistulas which follow. For the past eleven years he has raised and blocked off the facial skin from the growth beneath and then excised the carcinoma by diathermy through the mouth.

H. W. Meyer [35] stresses the necessity of thorough eradication of early basal cell carcinoma regardless of what method is employed. He cites the mutilating, difficult operations which must be done when the carcinoma is recurrent and illustrates his thesis with 4 examples.

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31. Davis, L., and Aries, J. L.: An Experimental Study upon the Prevention of Adhesions About Repaired Nerves and Tendons, Surgery **2**:876 (Dec.) 1937.

32. Duel, A. B., and Tickle, T. G.: Surgical Repair of Facial Nerve Paralyses, Ann. Otol., Rhin. & Laryng. **45**:3 (March) 1936.

33. New, G. B., and Figi, F. A.: Repair of Postoperative Defects Involving Lips and Cheeks Secondary to the Removal of Malignant Tumors, Surg., Gynec. & Obst. **62**:182 (Feb.) 1936.

34. Patterson, N.: Carcinoma of the Cheek, Brit. J. Surg. **25**:330 (Oct.) 1937.

35. Meyer, H. W.: Neglected and Recurrent Basal Cell Epitheliomas of the Face, Surg., Gynec. & Obst. **64**:675 (March) 1937.

The five year end results in the treatment of carcinoma of the tongue, lip and cheek as reported by Martin [36] from the Memorial Hospital leave one with the definite impression that there is still much to be desired in this field.

Plastic restorations following the treatment of carcinoma should, according to New and Figi,[33] be deferred until one year has passed. Excellent illustrative cases of the methods used in South America are reported by Prudente.[37] Neto [38] has advanced a new cheiloplasty, by which a large defect of the lower lip was repaired by transplanting a skin flap on a muscular tube taken off the biceps muscle. The author claims survival of the muscle and its adaptation to the lip, with functional activity.

Angiomas of the face, according to Figi,[39] are best treated with radium packs. This applies to the capillary angioma and to the cavernous angioma of the face in children. The cavernous angioma in adults is best treated by electrocoagulation.

As to the treatment of melanoma, Adair,[40] on the basis of 400 cases, advises the removal of moles which definitely contain black pigment. The means is not irradiation or electrocoagulation but well planned, carefully executed wide surgical excision.

Surgical infections, especially those due to streptococci, were favorably influenced by the new drug sulfanilamide. A vast literature is available on this effective therapeutic agent. Local infections of the face and jaws, especially when associated with necrotizing anaerobic organisms, responded well to the application of zinc peroxide, advocated by Meleney.[41]

The accumulated experience at the University of Minnesota on the treatment of actinomycosis led Wangensteen [42] to conclude that of the major methods of treatment, surgical intervention, the use of potassium iodide and irradiation, surgical intervention is of the greatest merit,

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36. Martin, H. E.: Five Year End-Results in Treatment of Carcinoma of Tongue, Lip and Cheek, Surg., Gynec. & Obst. **65**:793 (Dec.) 1937.

37. Prudente, A.: La chirurgie structive faciale appliquée aux operations pour cancer, Bull. et mém. Soc. d. chirurgiens de Paris, **28**:485 (Nov.) 1936.

38. Neto, J. R.: Nouvelle technique de transplantation musculaire appliquée à la cheiloplastie, Rev. de chir. structive, March 1936, p. 199.

39. Figi, F. A.: Treatment of Angioma of the Face, Arch. Otolaryng. **24**:271 (Sept.) 1936.

40. Adair, F. E.: Treatment of Melanoma: Report of Four Hundred Cases. Surg., Gynec. & Obst. **62**:406 (Feb.) 1936.

41. Meleney, F. L.: Treatment of Surgical Infections, Surg., Gynec. & Obst. **64**:387 (Feb.) 1937; Prophylactic and Active Use of Zinc Peroxide in Foul-Smelling Mouth and Neck Infections, Ann. Surg. **107**:32 (Jan.) 1938.

42. Wangensteen, O. H.: Role of Surgery in the Treatment of Actinomycosis. Ann. Surg. **104**:752 (Oct.) 1936.

particularly for faciocervical actinomycosis, for which to curet and to leave the wound open offer the best results.

Several papers on the dangerous areas of the face have appeared, but the dictum of treating these areas conservatively remains unchallenged. There is a tendency to employ small skin doses of roentgen rays. Roeder [43] has constructed a mechanical compression device to block off intracranial drainage from the face, and he cites 1 case in which it was used with favorable results.

PLASTIC OPERATIONS ON THE EAR

Most reconstructions of the external ear have failed from an esthetic point of view. Two articles may be mentioned for their account of results in this work.

Nattinger [10] reports successful grafting of maternal cartilage to the child. The graft includes the upper three quarters of the aural cartilage and extends well down to the auditory meatus, so as to include the concha. A pocket is made over the mastoid region large enough to accommodate the graft. After satisfactory healing has taken place, the pocket is reopened and converted into a flap composed of a thin layer of mastoid skin and a posterior layer of implanted cartilage. The mastoid wound and the posterior surface of the cartilage are covered with a thick razor graft. At this time a small tube is prepared in the preauricular region immediately in front of the tragus. After two weeks of healing, the lower end of the tube is freed from its base and swung around over the superior and outer margin of the ear to form a rolled helix.

Gillies [7] has used a similar technic and reports 9 successful cases. The fine results shown by his photographs are encouraging to workers in this field, which to date represented probably the most difficult and wholly unsatisfactory esthetic results.

A variation in the usual mastoidectomy incision is proposed by Dixon,[44] as it makes available a good blood supply and an adequate amount of periosteum for good closure. The incision is made parallel to the posterior border of the external ear, in the hairline, and a hidden scar, with little if any postauricular depression, is assured.

To prevent postoperative fistula, McNichols [45] extends the upper end of the usual mastoidectomy incision at right angles for about 2 inches (5 cm.). The flaps thus made available are freely undermined and then

43. Roeder, C. A.: Treatment of Infections of the Face by Prophylactic Venous Obstruction, Ann. Surg. **104**:1112 (Dec.) 1936.

44. Dixon, J.: A New Mastoidectomy Incision and Wound Suture, Ann. Otol., Rhin. & Laryng. **45**:75 (March) 1936.

45. McNichols, W. A.: Prevention of Post-Auricular Fistula by Periosteal and Subcutaneous Tissue Flaps, Ann. Otol., Rhin. & Laryng. **45**:475 (June) 1936.

split into an outer dermal and an inner subcutaneous-periosteal layer. The two layers are sutured separately, and as much of the inner layer as possible is turned into the mastoid cavity.

Postauricular fistulas are rather rare these days, but when they do occur they require a secondary plastic operation for closure. Ashley [46] uses a sliding split flap, whose inner flap is detached at its lower end and turned into the mastoid cavity to obliterate it and whose outer epidermosubcutaneous layer is bridged over the fistulous defect and closed by sutures.

Ersner and Myers [47] describe a variation of the pedicle flap for epithelization of the cavity created by radical mastoidectomy and report that it has given satisfactory results in 2 cases. A racket-shaped pedicle flap is taken from the lower end of the incision, which is extended into the neck. The flap is turned into the cavity to line the raw surface and held in place by a packing of plain gauze, which is led through the external auditory meatus. The anterior and posterior lips of the wound are closed over the flap, and after scarification of the cutaneous surface of the pedicle, the edges of the neck are closed over it.

The Bondy modification of the radical mastoidectomy is indicated in cases of cholesteatoma in which the perforation is limited to Shrapnell's area. Shambaugh [48] has added a refinement to the operation by lining the cavity with a Thiersch graft on a paraffin basket mold, as devised by Mosher. The basket remains in situ for from seven to ten days. In 2 cases of genuine cholesteatoma in the ear with the better hearing, surgical intervention was required. The Bondy modification of the radical mastoidectomy, with the use of primary skin grafting, resulted in a completely epithelized dry cavity in twenty-five and thirty-two days, respectively, and not only preserved but apparently improved the hearing in the ear on which the operation was performed.

Every surgeon sees at one time or another a person with prominent ears that need correction. In the older operation the posterior raw surface was stitched to the skin of the mastoid region, and resistant scar tissue was left in the mastoid furrow. In the newer method, as advocated by Graham,[49] an elliptical piece of skin and cartilage is removed from the posterior surface of the ear, and the edges of the cartilage and the skin are then closely approximated separately.

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46. Ashley, R. E.: Post-Auricular Fistula, Ann. Otol., Rhin. & Laryng. **46:** 477 (June) 1937.

47. Ersner, M. S., and Myers, D.: Variation of the Pedicle Flap for Epithelization of the Radical Mastoidectomy Cavity, Arch. Otolaryng. **23:**469 (April) 1936.

48. Shambaugh, G. E., Jr.: Primary Skin Graft in Modified (Bondy) Radical Mastoidectomy, Arch. Otolaryng. **23:**222 (Feb.) 1936.

49. Graham, H. B.: Plastic Surgery of the Ear, West. J. Surg. **44:**478 (Aug.) 1936.

Davis and Kitlowski [50] have laid down a standardized method for this operation, so that uniformly successful results can be expected. When the ear is held back manually in the desired position, that portion of the helix that is unfolded will reform itself to give the line of required reconstruction, and this is marked on the anterior surface of the ear with an alcoholic solution of brilliant green. A needle dipped into this solution is then used to make multiple punctures along this line about 1 cm. apart. The punctures on the posterior surface of the ear are connected, and then the ear is pressed against the skin of the scalp. The elliptical area enclosed between these two lines represents the area of skin to be removed. Excision of cartilage along the line of punctures is carried out if necessary. A special suture for forming an antihelix is used. No. 0 or no. 1 catgut is carried into the perichondrium on one side, about 0.5 cm. from the margin of the cartilage, and out close to the margin, then across the defect and into the perichondrium a similar distance from the margin, again emerging about 0.5 cm. from the defect. The skin is closed with on-end mattress sutures of horsehair. The postoperative dressing is important. All the crevices and dead space are neatly filled with small pieces of sea sponge. These are overlaid with a large sea sponge, which is fastened down with adhesive tape and fixed with a bandage. The best age for undergoing this operation seems to be about 4 or 5 years. Subsequent development of the ear proceeds normally.

PLASTIC OPERATIONS ON THE NOSE

Probably the most commonly injured part of the anatomy of man is the nose. According to Safian and Tamerin,[51] the nose is composed of six individual structures, and a simple diagnosis of fracture of the nose is of no value without a detailed analysis of the structures involved in the injury. Neglect of such injuries results in objectionable deformity, obstruction of the respiratory tract and possible involvement of the sinuses. Most fractures are amenable to reduction and immobilization. Certain types of injury are likely to make subsequent submucous resection and plastic operation necessary. In this group Safian places severe depressions of the bridge, some depressions of the septum, with saddle nose or dropped tip deformity, marked deviation of the cartilaginous bridge, occasional cases of protruding septum and, of course, markedly comminuted and compound fractures. The otolaryngologist must be able not only to recognize and treat these various types of fractures but to give a prognosis as to the outcome.

50. Davis, J. S., and Kitlowski, E. A.: Abnormal Prominence of the Ears: A New Method of Adjustment, Surgery **2**:835 (Dec.) 1937.

51. Safian, J., and Tamerin, J.: Recent Fractures of the Nose, Am. J. Surg. **31**:10 (Jan.) 1936.

The treatment of old unreduced nasal fractures is the subject of a paper by McIndoe.[26] Fomon [52] has published an illustrated review of lateral osteotomy through the vestibular incision for mobilization of the nose prior to replacement.

The external aspect of the nose is the subject of several papers. Telangiectasia of the nose was treated successfully by Biegeleisen [53] by the microinjection of a sclerosing solution of 30 per cent sodium chloride. Metzenbaum [54] has continued his studies on the etiology of septal displacements and has formulated some surgical principles on the basis of his experience. When the lower part of the septum is dislocated, until three weeks after the injury the part can usually be sprung back into the maxillary groove by manually rocking the nose or with the help of a forceps. Later, after a scar has formed and growth of the part in the dislocated position has occurred, replacement is best done by operation. Cohen [55] says that such injuries do not represent simply a dislocation of the lower border of the cartilage but that the posterior border, as well as the position of the septum in relation to the medial and lateral plates of the lower lateral cartilages, is disturbed, and he has devised an operation for their correction.

Reconstructions about the nasal tip may consist of procedures to replace defects or to alter the position or the size of this part of the nose. Straith [56] repairs defects of the tip of the ala by using nasolabial flaps, which leave an inconspicuous scar and provide excellent matched skin. For blemishes on the skin, Wolfe grafts from the upper eyelid or from the posterior aspect of the ear are suitable. When considerable tissue is needed, a cervical pedicle flap from the skin behind the ear can be transferred to the desired location on the face.

In correcting defects of the supporting structures of the nose, especially a broadly depressed cartilaginous bridge, Straith has extended Kazanjian's operation (in which the lateral wings of the alar cartilage are everted and sutured back to back to support the nasal tip) by including the upper lateral cartilage also. This procedure will often obviate the necessity of a cartilage transplant.

Roy [23] has simplified the Sheehan method of implanting a hinged cartilage support for a sinking nasal tip by using separate pieces.

52. Fomon, S.: Treatment of Old Unreduced Nasal Fractures, Ann. Surg. **104**:107 (July) 1936.

53. Biegeleisen, H. I.: Telangiectasia of the Nose: Treatment by Micro-Injection of Sclerosive Fluid, Ann. Otol., Rhin. & Laryng. **45**:416 (June) 1936.

54. Metzenbaum, M.: Dislocation of the Lower End of the Nasal Septal Cartilage, Arch. Otolaryng. **24**:78 (July) 1936.

55. Cohen, S.: Correction of the Displaced Septal Cartilage Especially in Children, Pennsylvania M. J. **40**:925 (Aug.) 1937.

56. Straith, C. L.: Reconstructions About the Nasal Tip, Surg., Gynec. & Obst. **62**:73 (June) 1936.

Wahl [57] has reduced the size of the tip by removing the lower anterior septal angle and placing an appropriate suture through the soft parts.

Some patients complain of obstruction of the respiratory tract and on examination with a nasal speculum reveal no intranasal abnormality. The condition is immediately corrected by raising the tip of the nose with the finger or with a speculum. Such functional stenosis is due to weakness and aspiration of the alar cartilages. Réthi [58] corrects these conditions by removing a small piece of the alar cartilage and allowing the resultant scar to stiffen the ala against aspiration. When the aspiration takes place near the free edge of the ala, a sickle-shaped excision of skin on the ala is carried out to raise this cartilage and keep it away from the septum.

True organic stenosis was studied in detail by O'Connor.[59] He expresses the opinion that there is usually sufficient skin coverage and cartilage support for the tip and alae but that the lack of mucosal lining for the nasal vestibule causes complete or partial closure of the nasal apertures. The simplest method of excision, as advocated by Réthi,[58] relieves the stenotic area but also shortens the nose. O'Connor [59] has devised an operation to remove the stenotic portion and replace it by skin grafting. An incision is made about the stenotic airway, starting at the base of the columella, proceeding to the tip of the nose and following the natural curve of the ala. Sharp dissection is carried into the nasal substance to create a pocket and to expose the medial crus of the ala, the superior ridge of the septum, the lateral crus of the ala, the lateral alar cartilage, the inferior ridge of the nasal process of the maxilla and the base of the pyriform opening of the maxilla. An impression is made of this pocket with dental compound (stent) and a Thiersch graft placed over it. This is sutured into the pocket. About two weeks later the tissue between the old stenosed airway and the newly grafted pocket is excised and serrated, so that the newly epithelized pocket can be swung into position and sutured to the remaining mucosa lining the base of the septum and the medial aspect of the nose.

Choanal atresia in newborn infants if unrecognized and untreated may be incompatible with life. Colver [60] has called attention to the necessity of early recognition of this condition and reports that removal

57. Wahl, S.: A Simple Method for Reducing the Abnormally Protruding Tip of the Nose, Rev.de chir. structive, June 1936, p. 315.

58. Réthi, A.: Treatment of Functional and Organic Stenosis of the Entrance of the Nose, Rév. de chir. structive, June 1937, p. 137.

59. O'Connor, G. B.: Operation for the Correction of Atresia and Stenosis of the Anterior Nares, Arch. Otolaryng. **25**:208 (Feb.) 1937.

60. Colver, B. N.: Congenital Choanal Atresia: Two Cases of Complete Bilateral Obstruction, Ann. Otol., Rhin. & Laryng. **46**:358 (June) 1937.

of the obstruction with a mastoid gouge through the nose is possible. Kearney [61] reports that if a secondary operation is to be avoided it is also necessary to remove the posterior edge of the vomer thoroughly.

Stenosis of the nasopharynx is usually due either to trauma or to syphilis. In the last patient seen by us the stenosis was secondary to a tonsillectomy and adenoidectomy. It is conceivable that in such patients wounds have a tendency to heal with the formation of a great deal of connective tissue, as they do in persons in whom keloids develop. Ordinarily perforation of the stenosed area, with dilation, causes in the end a denser stenotic mass. It is important that the opening produced shall be lined with epithelium to prevent adhesions and secondary stenosis. O'Connor [62] dissects a pocket bilaterally in the nasopharyngeal barrier and sutures into it a mold of modeling compound (stent) over which a Thiersch graft has been placed, with the raw surface out. A week or so later the stent is removed, the second stage of cutting the superior web attachment of the cicatrix is carried out and dilation treatments are begun. (One of the authors, R. H. I., recently performed this operation with excellent results.)

The use of mucosal lining of wounds to promote rapid healing and reduce cicatricial contractures has been applied to two intranasal operations. The external operation on the frontal, ethmoid and sphenoid sinuses, if it fails, usually does so because either the opening in the nose becomes inadequate or disease is left in the supraorbital extensions, with continuance of symptoms. McNaught [63] uses the Lothrop plan of removing the floor of both frontal sinuses, the interfrontal septum and the upper part of the anterior portion of the nasal septum to secure a large opening. To maintain it he raises a flap from the nasal septum, about 1.5 by 4 cm. being available. It is left attached to the ascending portion of the maxilla and turned into the opening of the frontal sinus. It is maintained for from five to seven days by rubber tubing until healing occurs.

Harris [64] suggests a submucous operation on the truly hypertrophied inferior turbinate in preference to the standard cauterization methods. He removes an elliptical section of the mucous membrane over the most

61. Kearney, H. L.: Congenital Bony Atresia of the Right Posterior Naris, Ann. Otol., Rhin. & Laryng. **45**:583 (June) 1936.

62. O'Connor, G. B.: Pharyngeal Reconstruction for Naso-Pharyngeal Stenosis: A New Operative Procedure, Ann. Otol., Rhin. & Laryng. **46**:376 (June) 1937.

63. McNaught, R. C.: Refinement of the External Fronto-Ethmosphenoid Operation: A New Naso-Frontal Pedicle Flap, Arch. Otolaryng. **23**:544 (May) 1936.

64. Harris, H. I.: Improved Technic for Submucous Turbinectomy, Ann. Otol., Rhin. & Laryng. **45**:481 (June) 1936.

prominent part of the inferior turbinate, separates off the adjacent periosteum, trims the bone and closes the flaps by a suture.

Various plastic operations which necessitate the introduction of cartilage to the bridge of the nose are best approached, according to Cohen,[65] through an incision which follows the lower border of the outer plate of the lower lateral cartilage. This line is easily exposed by pulling up on the edge of the outer vestibular margin with a fine retractor and everting the lower edge of the lateral cartilage by pressure with a finger.

PLASTIC OPERATIONS ON THE PALATE AND THE JAWS

Wardill and Whillis [66] had an opportunity to observe the mechanism of the movements of the soft palate in a patient who, as the result of an operation for carcinoma, had a wide opening through the lateral wall of the nose and orbit, through which the whole of the nasopharynx could be examined readily. The method of examination was visual observation from above and below, under direct and transmitted illumination. The palate was examined at rest, during speech, during deglutition and during blowing. From their observations the authors concluded:

> The movements of the palate in all phases of its activity are difficult to interpret and correlate with individual muscles. The diameters of the normal nasopharynx are very much smaller than might be imagined from the examination of a series of cases with unrepaired clefts of the palate. All movements are extremely speedy and, on superficial examination, little difference is observed between the nasal resonants and the explosive consonants. But in general it may be said that the greater the explosive effort required for the production of a sound, the greater is the elevation of the palate and the more firm the nasopharyngeal closure. It would seem that closure is considerably assisted by the heaping up of the mucosa by underlying muscles, and it can be imagined that, as has been found in clinical experience, complete nasopharyngeal closure is possible even with an almost completely immobile soft palate so long as the sling action of the levators remains intact. The tensor palati would appear to have little to do with the speech mechanism, its activity being strongest at the time of deglutition, and it is for this reason that it must be regarded as a muscle, the function of which is to propel the bolus over the back of the tongue.

Riemke,[67] of Copenhagen, reviews the history of operations for cleft palate, pointing out the disadvantages and ill results of the old Langenbeck, Brophy, Lane and other methods. He goes on to describe the more modern modifications: Rosenthal's pharyngoplasty, the retrotransposition operation of Halle and Ernst and Limberg and Dorrance and,

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65. Cohen, S.: A New Approach for Nasal Implants, to be published.

66. Wardill, W. E. M., and Whillis, J.: Movements of the Soft Palate with Special Reference to the Function of the Levator Palati Muscle, Surg., Gynec & Obst. **62**:836 (May) 1936.

67. Riemke, V.: Gaumenspaltenoperation, Hospitalstid. **78**:741 (July 9) 1935.

finally, Veau's operation, for which he predicts especially good results. An interesting feature of the literature has been the discussion between Axhausen, in Germany, and Veau, in France, regarding the principles involved in the treatment of cleft palate. Their papers are reviewed by Ivy [68] in his survey of the literature published during 1936 on congenital and acquired defects and deformities of the face and jaws.

The Dieffenbach-Warren operation for cleft palate is recommended as the standard operation of choice in America and is reviewed and illustrated step by step by Blair and Brown.[69]

It was formerly considered necessary to close a cleft palate before the characteristic speech associated with cleft palate had been established. The premise, according to Vaughan,[70] is incorrect, as such speech is dependent on the length of the palate and on whether when elevated it will function to prevent air from passing into the nose. Vaughan modifies the Langenbeck operation by using an M incision, which allows greater lengthening of the palate without the danger of an anterior perforation in cases in which the cleft extends for a considerable distance into the hard palate.

Wardill [71] carries out a preliminary pharyngoplasty for the purpose of narrowing the nasopharynx and making Passavant's ridge prominent. His palatal flaps differ from the usual type, as he uses a transversely oblique incision which commences at the posterior end of the hard palate and at the border of the cleft and runs obliquely forward and outward. The short palatal flaps allow the operator more liberty with the posterior palatine vessels, even making it possible for him to tie them off, if necessary, to free the mucoperiosteum.

Brown [72] preserves the posterior palatine vessels intact, using a two stage operation to elongate the palate and thus provide a good "shut off," which is so essential for correct speech.

An intratracheal device for inducing anesthesia in babies with harelip and cleft palate was produced and used successfully by Ayre.[73] Wardill concurs in his report.

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68. Ivy, R. H.: Congenital and Acquired Defects and Deformities of the Face and Jaws, Internat. Abstr. Surg. **64**:433 (May) 1937.

69. Blair, V. P., and Brown, J. B.: Dieffenbach-Warren Operation for Closure of Congenitally Cleft Palate, Internat. J. Orthodontia **22**:853 (Aug.) 1936.

70. Vaughan, H. S.: Surgical Repair of Cleft Palate with Special Reference to Lengthening the Soft Palate, Am. J. Surg. **31**:5 (Jan.) 1936.

71. Wardill, W. E. M.: Technique of Operation for Cleft Palate, Brit. J. Surg. **25**:117 (July) 1937.

72. Brown, J. B.: Elongation of the Partially Cleft Palate, Surg., Gynec. & Obst. **63**:768 (Dec.) 1936.

73. Ayre, B.: Anesthesia for Hare-Lip and Cleft Palate Operations on Babies. Brit. J. Surg. **25**:131 (July) 1937.

Padgett [74] reports a series of 114 cases of cleft palate in which operation had been performed unsuccessfully at an earlier date. The average number of unsuccessful operations per patient was 2.3. The principle of the Dieffenbach-Langenbeck operation was used in most of the cases in which there was little or no loss of tissue. When the palate appeared to be unusually short but sufficient tissue was still available for a good midline closure, the principle of uniting the posterior pillars, as advocated by Brophy, Blair and others, proved of value. Padgett describes in detail some of the procedures used for closure in cases in which there was definite loss of tissue. In operations on large defects of the soft palate a posterior pharyngeal flap was used to advantage. This type of flap permits a one stage operation that will successfully close a defective soft palate that could hardly be closed in any other way.

In certain cases a plastic operation either will be unsuccessful or is not feasible. Olinger and Axt [75] state that surgical prosthesis is indicated in such cases as follows:

(1) When quick painless results are desired by the patient

(2) When the deficiency is due to a disease that makes surgery a doubtful procedure

(3) When the defect is too extensive

(4) When the physical condition of the patient contraindicates the strain of a long series of plastic operations

(5) When a temporary device is desired preceding a series of plastic operations.

The treatment of fractures of the jaw has been fairly well standardized. The current methods of wiring have been reviewed by Doherty and Doherty.[76] Their remarks on the usual ineffectiveness of the Barton bandage in the treatment of mandibular fractures, as well as their conservative and successful treatment of condylar fractures of the mandible by simple immobilization of the teeth, are particularly worthy of reemphasis.

In the average case of prognathism, according to Pettit,[77] the defect is not a malrelationship in size between the maxilla and the mandible, but the lower jaw as a whole is set forward, chiefly by an abnormally obtuse angle of the mandible. The fundamental necessity is to create an additional angle farther back, so as to counteract the ill effects of

74. Padgett, E. C.: Repair of Cleft Palates Primarily Unsuccessfully Operated Upon, Surg., Gynec. & Obst. **63**:483 (Oct.) 1936.

75. Olinger, N. A., and Axt, E. F.: Surgical Prosthetics of Oral and Facial Defects, Am. J. Surg. **31**:24 (Jan.) 1936.

76. Doherty, J. L., and Doherty, J. A.: Fractures of the Jaws and Treatment, Surg., Gynec. & Obst. **64**:69 (Jan.) 1937.

77. Pettit, J. A.: Surgical Correction of Prognathism, J. Am. Dent. Assn. **24**:1837 (Nov.) 1937.

the obtuseness of the mandibular angle. He accomplishes this by resecting and repositioning the condylar neck, and he reports universal success in all cases.

Two other operations have been developed for protrusion of the mandible. In the first, a horizontal cut is made through the ramus of the mandible somewhere above the occlusal plane of the teeth. The body of the mandible is then pushed backward to the desired position and immobilized until consolidation of the bone is complete. The operation is simple in conception; its chief handicap is the occasional inability of the operator to control the upper fragments. It was first advocated by Babcock, and successful results are reported by Pichler, Bruhn, Kostecka and many others. Kazanjian [78] prefers the second method, that of removing a measured section from each side of the body of the mandible, preferably in the first molar region, pushing back the anterior fragment into its new position and immobilizing it with dental splints made previously for that purpose. Blair performed the first successful operation of this kind in 1898. Kazanjian had previously reported 5 cases and now adds 3 more.

Hensel [79] uses horizontal osteotomy and describes an operative approach to accomplish it under direct vision. For cutting the bone he has found that a dental motor with a contra-angle attachment, onto which a carrying shaft and a separating saw nine sixteenths of an inch in diameter are assembled, is a serviceable improvement on the chisel or the Gigli saw. He has carried out 14 operations with no complications and with entirely satisfactory results.

Bilateral retrusion of the mandible is discussed by Kazanjian and a report of 3 cases given. In 1 case Kazanjian divided the body of the mandible diagonally, pulling the chin forward and fixing it until union occurred in the new position. The chin was built out further in front by the insertion of an osteoperiosteal graft from the tibia. In a second case the bone was divided horizontally by dental burs on each side from just above the angle to the premolar region, and then the incision was carried up vertically to the alveolar ridge. This permitted the anterior part of the mandible to be brought forward and fastened by splints while the teeth were in occlusion. Rib cartilage was used later to add to the prominence of the chin. In a third case the lower jaw was built out to a satisfactory contour by placing a piece of costal cartilage in front of the symphysis.

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78. Kazanjian, V. H.: Surgical Correction of Deformities of the Jaws, Internat. J. Orthodontia **22**:259 (March) 1936.

79. Hensel, G. C.: Surgical Correction of Mandibular Protraction, Retraction and Fractures of the Ascending Rami, Internat. J. Orthodontia **23**:814 (Aug.) 1937.

Babcock [80] reports a new operation for retrusion of the mandible, in which he exposes the mandibular condyle through a temporal incision, displaces it forward and inserts a piece of costal cartilage between the condyle and the bony external auditory canal. Correction of the deformity and good articular movement are claimed for this procedure.

A review of this kind would not be complete without calling attention to the second edition of Sheehan's "Plastic Surgery of the Nose" and the second edition of Blair and Ivy's "Essentials of Oral Surgery." Both books have been rewritten and cover their respective fields in a thorough and systematic manner.

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80. Babcock, W. W.: Advancement of the Receding Lower Jaw, Ann. Surg. **106**:1105 (Dec.) 1937.

Abstracts from Current Literature

Ear

COMPLETE AVULSION OF THE SCALP AND LOSS OF THE RIGHT EAR. J. A. CAHILL and P. A. CAULFIELD, Surg., Gynec. & Obst. **66**:459 (Feb. 15) 1938.

The possibilities of pedicled tube grafting for the repair of complete avulsion of the scalp and loss of the right ear are described and illustrated in detail by means of photographs. Adequate protection of the cranium and the pericranium requires the use of full thickness skin. This is best obtained by pedicled tube grafts obtained either from the axilla or from the back.

MILLER, Philadelphia.

PATHOLOGIC CHANGES IN THE EAR IN LATE CONGENITAL SYPHILIS. O. MAYER and J. S. FRASER, J. Laryng. & Otol. **51**:683 (Nov.) 1936.

The extreme rarity of histologic examination in cases of deafness due to late congenital syphilis induced Mayer and Fraser to combine their anatomic material. They think that their investigation of the histologic and pathologic changes in the ear will render the clinical symptoms more readily understandable. The peculiarity of the ear trouble of congenital syphilis, apart from its connection with keratitis parenchymatosa and dental deformities, lies in the fact that it usually appears in apparently healthy persons, generally females, between the tenth and the twentieth year and that, once started, the deafness progresses rapidly, becoming pronounced overnight in some cases. Usually there is no pain, but the patient complains of tinnitus and frequently of giddiness. The tympanic membrane is perfectly normal or may be slightly infiltrated. The deafness is labyrinthine, but occasionally the middle ear may be slightly affected. Functional examination of the labyrinth frequently shows peculiar and inexplicable combinations of reactions. Although many cases have been studied clinically, the number in which there were anatomic and histologic examinations is small. The authors review the 5 cases that have been reported in the literature and then describe their own observations. They report first 3 cases in which late congenital syphilis was demonstrated clinically or anatomically and then 2 cases in which the diagnosis was made histologically only.

The first case is histologically interesting because of the extensive chronic gummatous osteomyelitis which cannot be ascribed to chronic suppurative otitis media or to tuberculosis but must be considered syphilitic. In the second case also the changes in the ear were characteristic of syphilis. According to the history in the third case, there can be no doubt that the condition was typical of congenital syphilis. Labyrinthine changes were the cause of the deafness. In the fourth case the middle ear was normal. The changes detected in the inner ear had been considered signs of meningococcic inflammation of the labyrinth, because the middle ear could not be looked on as the starting point of the trouble. This view was disproved by the condition of the semicircular canals and the bone of the labyrinthine capsule. The semicircular canals exhibited a most peculiar active chronic periostitis, characterized by being subperiosteal. Apart from this periostitis, the authors observed extensive chronic osteomyelitis of the labyrinthine capsule, with occasional nodules of lymphocytes, epithelioid cells and giant cells. Since cancellous bone was also formed, there was some resemblance to otosclerosis, but there are no "foci" in this case, the new bone being extremely irregular, as it usually is in inflammatory processes. The perivascular inflammatory infiltration, which was obvious everywhere, differentiated the condition at once from otosclerosis. Cerebral hemorrhage, which was the cause of death, points to

syphilis, and so do the articular changes. The case must be considered as one of late congenital syphilis. The gummatous inflammation at a limited portion of the anterior meatal wall is worthy of special mention as having induced the cholesteatoma in the external meatus. In the fifth case the diagnosis was osteomalacia, but the histologic picture of the ear undoubtedly points to late syphilis and probably to late congenital syphilis. EDITOR'S ABSTRACT.

PYRAMIDAL CHANGES IN THE ROENTGENOGRAM. L. KRAUS, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:15, 1936.

According to Kraus, there are a number of extratemporal processes which attack the petrosa and which in the beginning cause hardly any otologic symptoms. An epipharyngeal tumor, for instance, which erodes the basis of the temporal bone, does not cause symptoms until, in its destructive growth, it reaches the upper portion of the pyramidal apex and injures the gasserian ganglion and the abducens nerve. It has been found that by roentgen examination according to the method of Stenvers such processes can be located early; but, on the other hand, the roentgenograms are frequently given an erroneous interpretation. In order to avoid such errors, the author studied the histologic aspects of petrosal processes which had been roentgenologically examined. In a previous report he described his control examinations in 3 cases of petrositis, while in this report he analyzes 2 cases of epipharyngeal tumor, 1 case of tuberculous panotitis and 1 case of tumor of the cerebellopontile angle. In summarizing his observations on these 4 cases, he stresses the following points: 1. Destruction of the pyramid by malignant tumor of the epipharynx takes place from below, and roentgen examination reveals basal destruction first. 2. The median parts of the petrosa are destroyed earliest and most completely, and because of this the roentgenogram often reveals steplike destruction. 3. The anterior surface of the pyramid and the adjoining part of the bone are destroyed much earlier than the posterior portion, particularly the cortical portion of the posterior wall, which resists the longest. 4. The first three points are readily explained by the position of the primary tumor and by its growth tendency. 5. If the posterior pyramidal surface is intact, roentgen examination reveals a relatively clear outline of the apex, even if larger portions of the anterior part of the pyramid are missing. 6. If the posterior part of the cortex and the adjoining bone are lacking completely or partly, the roentgenogram shows a complete defect of the petrosa or circumscribed lighter areas. If the defect is localized around the porus acusticus internus, this process is dilated. 7. A lighter area in the central part of the apex in a case of malignant nasopharyngeal tumor may be the result of an inward growing of the tumor along the tube and its bony surroundings. An inflammatory peritubal process may thus perhaps be recognized by a lighter area in the center of the roentgenogram. 8. Atrophy of the entire petrosa may be caused by severe medullary changes in the form of active or passive hyperemia or by fibrous transformation of the marrow. 9. The temporary absence and the reappearance of the apex in the roentgenogram can be explained by the changing calcium content. The change in calcium content, too, is probably caused by active and passive hyperemia. This fact probably applies to inflammatory processes as well as to those that are accompanied by stasis. EDITOR'S ABSTRACT.

EXPERIENCES WITH RADICAL OPERATION THROUGH THE AUDITORY CANAL. B. KECHT, Monatschr. f. Ohrenh. **70**:1025 (Sept.) 1936.

Kecht thinks that of the many surgical interventions through the external auditory meatus which have been suggested instead of the external opening with the chisel, the radical method of Thiess deserves especial attention. He shows that if the correct technic is employed and the right cases are selected (those of cholesteatomatous suppuration with complete or almost complete inhibition of pneumatization), it deserves a place beside the external interventions and even

has a number of advantages. In the author's clinic the operation was performed 52 times on 49 patients. Follow-up examinations in 38 cases revealed complete cure in 23, secretion and the formation of granulations in 9 and persistence of the suppuration in 6. A second operation was necessary in 4 cases. The hearing was improved by the operation in 24 cases; it remained the same in 11 cases and was further impaired in 3. As advantages of the Thiess operation the author mentions the prevention of an external scar and with this the prevention of the complications arising from the retroauricular scar, greater preservation of the healthy bone and periosteum, lesser concussions from the chisel and thus less shock to the labyrinth, which in patients with cholesteatoma is usually constitutionally defective, better prospects for the later hearing capacity and shorter and simpler after-treatment.

EDITOR'S ABSTRACT.

PECULIAR SUBJECTIVE EAR SOUNDS. H. FREY, Monatschr. f. Ohrenh. **70**:1034 (Sept.) 1936.

Frey directs attention to a peculiar entotic ear sound, a low-toned humming which swells and then subsides. Occasionally it is continuous, but generally it recurs after shorter or longer intervals. Examination usually reveals normal aural conditions. Sometimes there are signs of mild pharyngotubal catarrh or catarrh of the tube and the middle ear. The author's observations seem to indicate that the sound is elicited by the muscles within the ear. Questioning revealed that many of the patients suffer simultaneously or at other times from blepharospasm. Moreover, if it is considered that many persons are capable of eliciting humming sounds in the ears by the voluntary contraction of muscles that are innervated by the facial nerve, the author thinks that it may be concluded that the described symptoms are the result of clonic, extremely slow contractions of the stapedius muscle. He suggests that contractions of the tensor tympani muscle may also cause such entotic sounds. To be sure, this has not been proved as yet. Regarding the therapy he says that local treatment (air douches, vibrations and so on) is usually ineffective. However, in view of the fact that spastic conditions of the muscles have been ascribed to a deficiency of fluids in the tissues, he recommends the intake of larger amounts of fluid. He also employs faradic current.

EDITOR'S ABSTRACT.

EXTRACT OF URINE OF PREGNANT WOMEN IN TREATMENT OF AURAL DISORDER. A. I. KOLOMIJTSCHENKO, Monatschr. f. Ohrenh. **70**:1038 (Sept.) 1936.

Kolomijtschenko tried a preparation of the urine of pregnant women in treating 27 patients with aural disorders. Because an endocrine factor seems likely in otosclerosis, he selected chiefly patients with this disorder. In 11 the otosclerosis had existed for over five years, and for these patients the extract was practically ineffective. Of the 6 patients in whom the otosclerosis had existed less than five years, 2 showed improvement. Of 7 patients with catarrhal otitis, only 1 was improved by the treatment. In 3 patients with neuritis of the auditory nerve, the treatment was entirely ineffective.

EDITOR'S ABSTRACT.

Pharynx

GLOSSITIS RHOMBICA MEDIANA. H. E. MARTIN and M. E. HOWE, Ann. Surg. **107**:39 (Jan.) 1938.

To the 42 cases in the literature, Martin and Howe have added 11 additional cases of glossitis rhombica mediana. The patients were referred to them as possibly having carcinoma of the tongue. Because of its unique position, this growth should be easily diagnosed and recognized as a benign process which requires no treatment or, at most, in a cancerophobic person, simple excision with immediate suture.

It is found chiefly in middle-aged men, occurring as a painless raised nonfurred, clear area in the midline of the tongue just anterior to the foramen caecum and the sulcus terminalis. It is believed to be of embryonic origin and may represent the belated appearance of the portion of the tongue derived from the tuberculum impar.

MILLER, Philadelphia.

INCIDENCE AND SIGNIFICANCE OF HEMOLYTIC STREPTOCOCCI IN THE THROATS OF CHILDREN. C. M. BURPEE, Arch. Pediat. **54**:470 (Aug.) 1937.

Cultures were taken from the throats of 148 white and 152 Negro children and 63 nurses and doctors at intervals of two weeks over a period of one year. The average incidence of carriers of Streptococcus haemolyticus was 11.1 per cent among the patients and 6 per cent among the members of the staff. There was a higher incidence of positive cultures for patients with tonsils. The incidence of positive cultures was higher for white than for Negro patients.

The incidence of Str. haemolyticus in patients in a pediatric ward can be controlled within certain limits. The incidence of Str. haemolyticus in the throats of the members of the staff was not definitely related to that in the throats of the patients.

The incidence of Str. haemolyticus in the pediatric ward showed seasonal variations. The percentage of carriers was highest in November, December, February and July, and this observation seemed to be independent of the illnesses prevailing at these times. Burgin and Higgins found the greatest incidence in November and March.

ORR, Buffalo, N. Y. [AM. J. DIS. CHILD.]

RADICAL TREATMENT OF PERITONSILLAR ABSCESS. W. S. THACKER NEVILLE, J. Laryng. & Otol. **51**:650 (Oct.) 1936.

Thacker Neville says that the early removal of tonsils in order to drain a peritonsillar abscess when the ordinary palliative incision fails, when the abscess is situated between the middle third or the lower third and the lateral wall of the pharynx or in ordinary cases to shorten the length of the illness is not widely practiced. He adopted this procedure after reading Canuyt's publications. The author states that he removed the tonsils in cases of peritonsillar abscess with paraldehyde followed by ether as anesthetics or with ether alone. He prefers ether alone, as the recovery is quicker. He points out that peritonsillar abscesses are not always situated at the upper pole of the tonsil, as the infection may gain entry through any crypt, but may be situated anywhere between the tonsil and the lateral pharyngeal wall. While a long-standing abscess in the upper third of the tonsillar fossa is easy to drain through a palatal incision, an abscess in its early stages or an abscess in the middle or lower part of the tonsillar fossa is impossible to drain through the usual incision. Canuyt discovers the presence of an obscure abscess by puncturing the anterior faucial pillar with a needle attached to a syringe. He makes a series of punctures and so explores the upper two thirds of the fossa. Such a series of punctures would prevent an unnecessary incision and would also enable one to diagnose an abscess before the palate was affected. Canuyt is not alone in recommending exploratory punctures, as Key-Aberg considered that if the clinical signs point to a peritonsillar abscess and if three exploratory punctures fail to reveal the abscess, one ought to do an immediate tonsillectomy, as the negative results of punctures point to the presence of a deep-seated abscess. After discussing the recommendations of these and other authorities, the author reports the histories of a number of patients treated by himself. He concludes that the treatment for a peritonsillar abscess should consist of admission of the patient to a hospital, wide incision, producing dislocation of the upper pole of the tonsil, and total removal after the lapse of a few days, when the temperature has become normal; or it may, in certain cases in which the general symptoms are mild or the abscess is on the lower pole, consist of opening of the abscess followed by aspiration and then immediate removal.

EDITOR'S ABSTRACT.

Larynx

TRACHEAL STENOSIS: TWO CASES. A. CAMPBELL, J. Laryng. & Otol. **51**:644 (Oct.) 1936.

The first case reported by Campbell was one of complete tracheal stenosis of traumatic origin. In the second pulmonary tuberculosis existed. However, the author emphasizes that there was never anything in the larynx to indicate tuberculosis, and since the condition of the patient's nose strongly suggested syphilis, he considers a syphilitic cause possible. In both cases the tracheal stenosis was finally counteracted by a laryngotracheotomy. The author points out that laryngotracheotomy was first done for laryngeal stenosis by Heryng in 1894. Chevalier Jackson performed it in 1900 and reported 5 cases in which it was successful. In 1906 Killian demonstrated that rubber tubing is most effective in softening and absorbing the obstructive cicatricial tissue. The author did the operation as it was described by Jackson, who recognizes the value of Killian's discovery of the effect of rubber tubing on cicatricial tissue. The object of the operation is to get rid of cicatricial tissue and to cover the newly formed lumen of the trachea, first with small firm granulations and then with epithelium.

EDITOR'S ABSTRACT.

SIGNIFICANCE OF TECHNIC OF IRRADIATION AND OF TISSUE STRUCTURE FOR EFFICACY OF ROENTGENOTHERAPY OF LARYNGEAL PAPILLOMAS. J. KOCH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:83, 1936.

Koch reports his observations with roentgenotherapy in 22 cases of laryngeal papillomatosis. He classifies the cases in three groups: (1) those in which roentgenotherapy without previous surgical intervention was used, (2) those in which irradiation was employed on account of relapse after several unsuccessful operations and (3) those in which irradiation was used after total surgical removal of the papillomas. On the basis of his observations he advises that favorably located papillomas should be removed by operation as completely as possible and the site subjected to postoperative irradiation. Eight of the author's cases seem to prove that irradiation counteracts the proliferative capacity of the mucous membrane and thus prevents recurrences. If the conditions are unfavorable for total removal by surgical means, primary irradiation is to be preferred to inadequate surgical treatment. Regarding the technic of irradiation, the author says that he always used hard rays and that continuous fractional doses are superior to distribution of the total dose over several series. Since the laryngeal papilloma is not sensitive to rays, the total dose should not be too low. Histologic studies convinced the author that differences in tissue structure imply differences in sensitivity to rays. Whereas papillomas with a dense structure of connective tissue and with only a few vascular but many cellular elements responded well to irradiation, those which had a looser structure, were highly vascularized and were deficient in cells had considerable resistance. This implies that in determining the technic of irradiation it is not sufficient to know that the growth is a papilloma; the histologic structure should likewise be taken into consideration.

EDITOR'S ABSTRACT.

UNUSUAL VOCAL DISORDER FOLLOWING STRUMECTOMY. R. SOKOLOWSKY, Monatschr. f. Ohrenh. **70**:1170 (Oct.) 1936.

Sokolowsky discusses the history of a woman singer (mezzosoprano) who underwent a thyroidectomy for goiter. The postoperative course was uneventful, but when the range of the voice was tested it was found that the upper tones of the middle register and all those of the head register were missing. The speaking voice was unimpaired. A review of the literature suggested that the vocal disturbance might be the result of surgical impairment of the external laryngeal muscles. In the case under consideration, three muscles seemed to be of interest, the sternohyoid, the sternothyroid and the omohyoid. The author gained the

impression that the severing of the omohyoid was probably of no importance but that the vocal disturbance was the result of injuries to the sternothyroid and the sternohyoid muscle. These two muscles apparently play an important role in vocal activity, particularly in the formation of the higher tones. That such vocal disturbances are not more often observed after thyroidectomy and the involved severing of the two muscles is probably due to the fact that they are detected only in professional singers, for the speaking voice is not impaired. Moreover, if the severed muscles are properly taken care of, the vocal impairment may be avoided.

EDITOR'S ABSTRACT.

Nose

INFECTIONS OF THE PARANASAL SINUSES OF DENTAL ORIGIN. J. J. SHEA, Surg., Gynec. & Obst. **66**:408 (Feb. 15) 1938.

In reviewing the histories of 32 radical operations on the antrum, Shea says that 2 were done because of malignant processes, 2 followed fractures resulting from automobile accidents, 7 were for dental fistulas following the extraction of teeth and 9 were for hypertrophic membranes or empyema as part of pansinusitis; 12 of the antrums contained one or more polyps. In 6 cases the polyps were considered of dental origin because of the site of their attachment, and in the other 6 a bilateral operation on the antrums was required. These interrelated conditions, as well as accidental perforations of the antrum during the extraction of teeth, dental cysts (the radicular and the follicular cyst, adamantinoma and dermoids) which impinge on and often displace the sinus considerably, and aberrant teeth, which may be found in the sinuses, where they act as foreign bodies, require an appreciation of both the dental and the otorhinologic aspects for their successful treatment.

MILLER, Philadelphia.

INVISIBLE SCARS IN EXTERNAL AND ETHMOIDAL OPERATIONS. A. L. YATES, J. Laryng. & Otol. **51**:715 (Nov.) 1936.

Yates defines as an invisible scar one which cannot be detected in daylight at a distance of 2 feet (0.6 meter). A visible scar he defines as one which can be seen at 2 feet but not at 20 feet (10 meters) and an unsightly scar as one which can be seen at more than 20 feet. The author found that in certain areas an unsightly scar is apt to follow a frontoethmoidal operation and that in certain areas the scar will generally be invisible or visible only at a short distance. An invisible scar can be obtained with the greatest degree of certainty if the incision is confined to the region of the eyebrow. The eyebrow can be mobilized and brought down to the lateral margin of the nose so as to effect the removal of the bone in the Kisch position. The exposure thus obtained is inferior to that obtained by the Kisch incision, and a choice must be made in individual cases between the maximum of invisibility of the scar and the maximum degree of surgical exposure. The choice in many cases is determined by the degree of osteitis present.

EDITOR'S ABSTRACT.

RADIOTHERAPY OF BASAL FIBROIDS. J. KOCH and G. EIGLER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:1, 1936.

After a review of the literature on the treatment of basal fibroids, Koch and Eigler report cases that were observed at the clinic in Halle, Germany. In view of the fact that when there is considerable extension of the basal fibroid operative removal may involve danger to the life of the patient, all basal fibroids at the Halle clinic were subjected to irradiation. In 2 cases the fibroid had already spread to the adjoining nasal sinuses, whereas in the other 2 the fibroid was smaller and restricted to the nasopharynx. In all except 1 of the cases treatment was by a combination of roentgen and radium rays. In the 1 exception only larding with radium was employed. The irradiation was well tolerated, in spite

of the fact that in some cases large doses of roentgen rays had to be applied on account of the considerable extension of the growth. The effect of the radium rays was especially favorable, and for this reason the authors recommend the use of radium whenever the growth can be reached easily. Of the 6 patients, 5 were completely cured and the sixth is still undergoing treatment. The dose of rays has to be adapted to the size of the tumor and to the age of the patient. The histoloic aspects of the exploratory excision cannot be taken as a basis for the dosage, because the cell structure varies in the different parts of the basal fibroid. The mucous membrane was not noticeably impaired by the treatment with rays. The authors conclude that for the treatment of large basal fibroids irradiation is the method of choice.

EDITOR'S ABSTRACT.

DEVELOPMENTAL CHANGES IN THE MUCOUS GLANDS AND THE CAVERNOUS TISSUES IN THE MUCOSA OF THE RESPIRATORY PORTIONS OF NOSE. L. A. ZARITZKY, Monatschr. f. Ohrenh. **70**:1057 (Sept.) 1936.

Zaritzky describes postmortem histologic studies on the nasal mucosa of children and adults of various age groups. He observed that the layer of cavernous tissue of the mucous membrane is much thinner in nurslings than in adults. In nurslings this tissue is almost evenly distributed over the entire lower concha, whereas in adults it is only on the ends of the concha. The connective tissues, the strands and the fiber bundles which make up the cavernous tissue are much more delicate in young children than in adults and are less readily stained with Van Gieson's stain. The nasal mucous membrane of young children has only few mucous glands. Not until the fifth year of life do these glands reach their full development. After the age of 30, the functional activity of some of the mucous glands occasionally decreases, and after the age of 40 the decrease is a normal manifestation.

EDITOR'S ABSTRACT.

Miscellaneous

FUNDAMENTALS OF THE PROBLEM OF PNEUMATIZATION. A. ECKERT-MÖBIUS, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:43, 1936.

Eckert-Möbius demonstrates that the various forms of pneumatization which develop under normal and pathologic conditions in the temporal bone and in the different bones of the facial part of the skull can be explained largely as the result of the dysfunctional-dystrophic and the functional-trophic metabolic actions that take place in the growing bone. The form-giving power of the pneumatizing epithelium doubtless plays a decisive part in the "primary pneumatization" that takes place in the as yet little differentiated embryonal tissue. However, in the "secondary pneumatization," that is, in the bone pneumatization as such, it is of considerably less importance than the dystrophic and the functional-trophic processes that are involved in the growth of the bone. The building and decomposition processes in the bone produce, by the decomposition of the dystrophic regions of bone, the requirements for a further extension of the sinusal mucous membrane. The author is convinced that the chief cause of the pneumatization of the mastoid process and of the facial bones is to be found within the bone itself and that in the last analysis the pneumatization of certain bones of the skull is due to the fact that they adjoin hollow spaces which contain air.

EDITOR'S ABSTRACT.

SURGICAL TREATMENT OF PULSION DIVERTICULUM OF THE ESOPHAGUS. G. HOFER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:54, 1936.

After reviewing the early literature on the pulsion diverticula of the esophagus and after mentioning the two theories as to cause (mechanical and congenital), Hofer points out that the clinical manifestations differ greatly. In discussing the diagnosis, he says that it can often be made on the basis of the anamnesis and that examination with the esophagoscope must be conducted with great care. In

taking up the treatment, he mentions and discusses the following methods: invagination according to Girard, ligation of the diverticulum according to Goldmann and von Beck, exteriorization according to Liebl-Bogoras, fixation according to König-Schmid and extirpation of the diverticulum in one or two stages. He considers the last method the most radical and reliable one and thinks that it can be carried out in one stage, provided certain precautions are taken. He shows that the success of the operation is largely dependent on the careful preparation of the patient, in which thorough cleansing of the diverticular sac is of primary importance, for if this is not accomplished there is danger of a secondary infection of the wound even if the diverticulum is extirpated with the greatest care. He further demonstrates that simple and brief drainage of the cervical wound after the extirpation is not sufficient, for there is danger of sepsis, of arrosion hemorrhage from the large cervical vessels and of gravitation of the suppuration toward the mediastinal space. To avoid these complications the author recommends the introduction of a tight tampon of iodoform gauze at the lowest end of the wound. This protection of the mediastinum by tamponade has to be continued for a sufficient length of time. Its duration is dependent on the closure of the esophageal wound. If the healing takes place by primary intention, the tampon remains only a short time, but if a fistula forms it may have to remain for three weeks. Besides protecting the mediastinum, it is necessary to give careful attention to the esophageal wound. The author advises that attempts at swallowing be made while the external wound is still open, so that it can be determined whether the esophagus is free from leaks. This prevents later complications. The author describes 4 cases in which the protection of the mediastinal space could be discontinued from ten to fourteen days after the operation and the drain could be removed from the wound from eighteen to twenty-eight days after cure.

EDITOR'S ABSTRACT.

THERAPEUTIC USE OF IODIZED POPPY SEED OIL IN TREATMENT OF CEREBRAL AND CEREBELLAR ABSCESSES. B. KARBOWSKI, Monatschr. f. Ohrenh. **70**:1044 (Sept.) 1936.

The fact that in cases of pulmonary abscess the introduction of iodized poppy seed oil is often followed by improvement or cure induced Karbowski to try this substance in treating abscess of the brain when the postoperative course was not satisfactory. He reports 2 cases. He ascribes the favorable influence of iodized poppy seed oil on the postoperative course of abscesses of the brain to the strongly disinfecting action of the substance, with its 40 per cent iodine content, and to the favorable effect of the fat on ulcerating surfaces. Moreover, the high specific gravity of the substance facilitates the evacuation of the abscess, because the suppurating exudate gathers above it. The 2 cases observed by the author indicate that fillings of iodized poppy seed oil are well tolerated. The resorption of the substance is extremely slow.

EDITOR'S ABSTRACT.

EXOPHTHALMIC GOITER AND VESTIBULAR DISORDERS. B. VON FIALOVSZKY, Monatschr. f. Ohrenh. **70**:1050 (Sept.) 1936.

Von Fialovszky describes the clinical history of a man, aged 40, in whom a vestibular disturbance developed simultaneously with the symptoms of exophthalmic goiter. Later, as the symptoms of the exophthalmic goiter subsided, the vestibular symptoms disappeared likewise. In estimating this case, the author considers three possible explanations for the concurrence of the exophthalmic goiter with the vestibular symptoms: (1) an accidental concurrence, (2) a common cause and (3) the exophthalmic goiter as the cause of the vestibular symptoms. Regarding the third possibility, he says that exophthalmic goiter might have elicited the vestibular symptoms either by way of a thyrotoxic action or by way of circulatory disturbances. In view of the fact that the cochlear function was not impaired, the author excludes a peripheral and assumes a central lesion and suggests that the latter might be the result of circulatory disturbances.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

DR. WALTER H. THEOBALD, M.D., *President*

Regular Monthly Meeting, Jan. 3, 1938

PRESENTATION OF CASES

DR. J. R. LINDSAY: Five clinical cases were reported before dinner. Two of these, the case in which movements of the stapes were visible and that in which cochlear function was still present and facial movements had returned after an operation on the labyrinth (the canals and the vestibule were opened posteriorly and the promontory removed between the oval and the round window) and decompression of the facial canal, will not be discussed unless questions are asked.

The 2 cases in which petrositis was cured will be reported later.

In the fifth case, reported as an instance of laryngeal vertigo, the patient had been ill for eight months. In May he began to have spells of coughing. During the more severe attacks he would fall down unconscious, and in fifteen or twenty seconds he would get up apparently quite normal. He broke his fibula in one fall. He was observed on some of these occasions, and on at least one of them he had epileptiform movements. He fell down only when the spell of coughing was severe. For a while he fell as often as twenty times a day, but the attacks became infrequent. Neither the local examination nor the general physical and the neurologic examination revealed the cause of the coughing or the falling. The attacks could not be induced by the breathing of carbon dioxide, by rebreathing or by pressure on the carotid sinus.

The name laryngeal vertigo is used here because it has been used for such conditions in the literature. There is no vertigo.

Attention is drawn to an article by Dr. J. Gordon Wilson (Laryngeal Vertigo: Its Relation to Cataplexy, *Arch. Otolaryng.* **15**:534 [April] 1932) in which he pointed out the similarity of laryngeal vertigo to cataplexy.

DR. S. A. FRIEDBERG: I think Dr. Galloway's modesty kept him from talking about these cases. I shall briefly outline the histories. Dr. Galloway was the surgeon in each instance, and his judgment was undoubtedly responsible for the excellent results. The first patient was an 11 year old boy who had a complete mastoidectomy after five weeks of persistent suppurative otitis media. He was discharged, practically well, in ten days. He returned a week later with profuse recurrent discharge from the middle ear and complaining of severe headache on the left side and pain around the eye. His temperature had been 101 F. each evening. A roentgenogram showed some involvement of the petrous apex. The suppuration was mostly from the mastoid wound. The wound was reopened; an area of softened bone was found behind the posterior semicircular canal, which was followed toward the petrosa, and frank pus was encountered. The patient made a complete recovery. The operation was performed on the basis of severe headache, pain in the eye, elevation of the temperature, recurrence of drainage from the mastoid wound and the roentgen findings.

In the second case the condition was very complicated. The boy was brought into the Cook County Hospital in a diabetic coma, with a history of having had a tooth extracted two weeks previously. There were frank signs of meningitis; he had paralysis from the second through the eighth cranial nerve on the right side. There were signs of diabetic acidosis in addition to the basilar meningitis. The patient began to recover clinically when the acidosis was controlled. In the course of four or five weeks, sequestration of the hard palate and part of the sphenoid took place. After approximately five weeks he complained of excruciat-

ing headache and pain in the right eye. It was felt that there was some involvement of the petrous apex, probably secondary to osteomyelitis of the sphenoid bone. Dr. Galloway explored the mastoid and encountered a large abscess in the anterior part of the petrosa. The boy was relieved of headache. At the present time there is a large defect in the palatosphenoid area. The diabetes is controlled. There is a fistula into the floor of the sphenoid from the petrous apex, demonstrable by irrigation with alcohol via the middle ear. The status of the involved nerves remains the same. There is slight drainage from the middle ear. Ligation of the carotid artery was considered because of the proximity of this vessel to the large sphenoid defect, but so far the patient has progressed satisfactorily, and the lesion is quite clean.

DR. T. C. GALLOWAY: With reference to the second case mentioned by Dr. Friedberg, it was thought for several reasons that the condition might be osteomyelitis of the petrosa arising by direct extension from the nasopharynx. In the first place, the third, fourth, fifth, sixth and seventh nerves were involved before the ear had drained. The palate was involved first. It is significant that at operation the posterior cells of the mastoid were seen to be nearly normal and the anterior-superior group more involved. The petrosa was found to be little more than a shell filled with thick pus. The process may have gone up through the tube, but quite likely the areas of sequestration represented the true pathway.

SUPPURATION OF THE PETROUS PYRAMID. DR. J. R. LINDSAY.

Pathologic material from 8 cases of meningitis of otitic origin was presented, and 3 clinical cases were reported in which suppuration of the petrous apex was proved and successfully treated.

The presentation of pathologic material included photomicrographs from 7 cases of suppuration of the petrous pyramid and 1 case of labyrinthitis with abscess of the saccus endolymphaticus, which were collected over a period of seven years. These 8 cases were selected from a group of 28 cases of meningitis of various types, in 15 of which there was associated otitis media.

Some of the important features illustrated by the pathologic examination were as follows:

Macroscopic appearances at autopsy usually did not give sufficient information to indicate the pathway of extension to the meninges. In order to get the exact information, which is necessary if an adequate method of treatment is to be arrived at, the histologic examination is essential.

In 5 of 7 cases of abscess in the petrous apex (including 3 clinical cases), there was no fistula to the middle ear or the mastoid, while in 2 cases a microscopic tract of suppuration was demonstrable. These cases show that the abscess in the apex frequently cannot be reached from the mastoid, that it is frequently, if not usually, of the closed type and that it may rupture in any one of several directions. The infecting organism was the hemolytic streptococcus in most cases, but in 3 cases pneumococci of type III were observed and in 1 pneumococci type I.

In 1 of the cases in which pneumococci of type III were observed, there was a large abscess in the apex, with rupture of the middle fossa and meningitis within two weeks of the onset. In the other cases of pneumococcic infection the disease was more prolonged and was accompanied by little fever until the meningeal infection developed.

In the cases of streptococcic infection, either the course was sometimes prolonged, with little or no fever, or it was stormy and febrile from the onset. A septic temperature occurred only in the cases of streptococcic infection and usually indicated thrombosis of a venous sinus. With suppuration of the apex there may be involvement of the carotid venous plexus or of the cavernous and the petrosal sinuses before the jugular bulb or the lateral sinus is involved. The most essential feature of the treatment in such cases is drainage of the suppurating focus in the bone.

In every case the infection had progressed into the pyramid by way of pneumatic spaces, with production of an area of suppuration and absorption of bone, which later ruptured through the cortex to the meninges. The bone marrow showed marked resistance to the infection. Diffuse fibrosis occurred in the marrow spaces in most cases, affecting gray and red marrow alike, with some osteogenesis adjacent to the suppurating focus. This reaction in the marrow is not true osteomyelitis, and it provides an adequate defense against invasion and destruction by the infection.

Bilateral acute osteomyelitis occurred in 1 case as a terminal process.

The most constant symptom was pain, the location of which in the early stages was an important indication as to the site of the lesion. Aural discharge was absent in 2 of the cases of apicitis, and palsy of the sixth nerve was present in 3. A Babinski sign on the side opposite the involvement was an early sign in 2 cases in which cure took place, indicating that a local meningeal reaction was present in the middle fossa, although the spinal fluid was normal.

The roentgenographic findings are a valuable aid to diagnosis. I have found the axial view most useful, although the occiput-down and the Stenver views are used as a matter of routine.

These cases demonstrate that suppuration of the petrous pyramid should be diagnosed while the infection is still in the intrapetrosal stage. A safe means of procuring drainage from a "closed" abscess is a necessity. If surgical intervention is delayed until rupture of the cortex takes place, meningitis may be unpreventable. The intrapetrosal method of draining an abscess in the apex from the mouth of the eustachian tube was used in 4 cases with a satisfactory result.

DISCUSSION

Dr. E. W. Hagens: About the only way I can discuss this paper is to compare and contrast Dr. Lindsay's experiences with my own. His presentation is difficult to discuss, because his findings speak for themselves. I think it is appreciated that the petrous bone varies a great deal in its anatomy. In the newborn it consists of an outer, or periosteal, layer, an inner, or endosteal, layer and between the two a layer which consists of a great deal of marrow. The layer of marrow changes greatly as time goes on. In some instances the spaces become smaller and the bone becomes solid. In other cases considerable marrow remains, particularly in the apex. In still other cases pneumatization takes place, as in the mastoid, and the petrous bone varies from being solid to being extremely pneumatized. I think that these facts must be borne in mind when one evaluates clinical cases.

I followed a number of children at the County Hospital between the ages of 6 months and 14 years. Twelve died of meningitis. I did not know whether they had petrositis or not, but they had otitis media. I thought it would be interesting to know whether the petrous bone was involved, because clinically one has to decide this matter in order that one may know whether to operate or not and how conservative or how radical the procedure should be. Six of the 12 children were operated on, and the others were not. Then the question was: Had petrositis been missed in any of the cases? Had it been present without breaking through and causing meningitis?

My experience differs somewhat from Dr. Lindsay's, but if he were to include the 15 cases of petrositis, my figures would coincide better with this. It seems to me that infection spreads mainly through the pneumatized spaces and then through the marrow and that last of all it will erode bone. A hard petrous bone, however, will resist infection. I found osteomyelitis in the petrous apex in 3 cases; the area of erosion was so large that one could hardly recognize the marrow cavity. The ages of the children were 3, 4 and 5 years, respectively. It has often been stated that when little or no pus is found one is dealing with a thrombotic phenomenon and that meningitis develops by extension through the thrombosed vessels. I do know that this phenomenon can occur, but I did not

see it in the 12 cases I studied. In 1 the pathologist was sure he would find it, because there were thrombi throughout the body; yet the sections did not reveal any thrombi. I am speaking only of the petrous bone. I also found labyrinthitis in most of these cases. It seemed to be secondary to the meningitis. In only 1 case could I find a spread through the round window, and here no perforation was seen, just a thinning of the membrane of the window. I believe the labyrinth is well insulated, so that infection does not travel as readily as one might expect.

Regarding bacteria, Dr. Lindsay mentioned that in some of his cases he isolated streptococci. In my series there were streptococci in 4, pneumococci in 6 (the pneumococci were definitely of type III in 1), influenza bacilli in 1 and a mixture of organisms in another. Since 1918 physicians have talked about influenzal otitis media. It was interesting to me to see hemorrhage through the labyrinth, for the picture was entirely different from that in the other bones; it looks as though with influenza the picture is different throughout the ear. I have enjoyed Dr. Lindsay's paper, and I think that after a summation of all the series along this line the variations possible in petrositis will finally be appreciated and the condition observed more intelligently and treated surgically.

DR. W. E. GROVE: I have had considerable trouble in getting roentgenograms, and I should like to know how Dr. Lindsay produced the excellent pictures he showed.

DR. SAMUEL J. PEARLMAN: Dr. Lindsay's paper is admirable. I could not help thinking as he spoke how much there remains for me, at least, before the picture of petrositis will be altogether clear.

My memory goes back to a case of some years ago which illustrates, I think, the general attitude at that time. A youngster had otitis media for over two weeks and began to complain of terrific headache. Impelled by a fear of impending meningitis, the surgeon operated, and within a day or two paralysis of the external rectus muscle developed. I remember how happy I was then because my associates and I felt that our troubles would be over. We felt that the condition was, in the parlance of the day, Gradenigo's syndrome in association with otitis media, and we all knew that the majority of the patients with that condition get well without any further intervention. We also knew that in a certain number of them meningitis develops, but we thought that we could not do much to prevent it.

On the other hand, a few weeks ago a youngster entered the Cook County Hospital with frank meningitis. A hemolytic streptococcus was recovered from the spinal fluid. He had unilateral otitis media of about eight days' standing, with an intact membrana tympani, and a paracentesis was done at once. There had not been any discharge previously. Sulfanilamide was administered, and the child rapidly improved. The mastoid process was not operated on. In former days such an operation and drainage of the spinal fluid would have been the only things to do.

The new chemotherapy is bringing such marked changes that physicians seem to be facing what may be a rather important revision of operative indications, particularly with regard to otitis media complicated by meningitis. Mastoiditis, of course, has indications all its own. Furthermore, one cannot help speculating about the pathologic changes in these troublesome cases and asking the nature of a process which yields so quickly to therapy with drugs. Surely a destructive lesion in a bone cannot heal in from forty-eight to seventy-two hours. Otolaryngologists may look forward, I hope, with a great deal of optimism and with expectation to a rapid disappearance of the confusion of today, in view of such work as Dr. Lindsay has reported.

DR. T. C. GALLOWAY: I want to point out one value of Dr. Lindsay's work. He has visualized the pathologic process. He has pointed out the proper approach in each case, and I think that if his approach is followed one will not only make

a positive diagnosis of petrositis in many cases but will be able to make a specific diagnosis as to the part of the petrosa that is involved and therefore get indications as to the operative procedure.

Dr. Joseph C. Beck: As an old-timer, with considerable experience in operating on mastoids in public institutions and in private practice, up to this day I have not done an operation on the petrous pyramid. I recall few cases in which meningitis followed a mastoid operation, and when this work on the petrous pyramid was first brought out and surgical intervention enthusiastically spoken of, I was unhappy about the whole thing and said to myself, "Now we will have a holocaust of deaths from this enthusiasm." After going through the literature and checking the cases reported by Kopetsky and others, including 2 cases in which operation was successfully performed by my associate Dr. M. R. Guttman, I am glad to say that I have lived long enough to change my mind.

As Dr. Pearlman and Dr. Galloway mentioned, the two physicians who presented the paper and those who discussed it rounded out the whole picture fairly well. Only time will tell exactly when and how to operate. Merely that one surgeon has not seen any deaths from meningitis after a mastoid operation is not a criterion. Many cases are on record in which the diagnosis was made properly and the operation performed in the proper way. I do not think of the boring operation when I say this, but I think of the uncapping of the petrous pyramid through an opening that can be seen at the base of the skull. Some of the cases reported here showed clearly that somebody was just too late and let the abscess formation go on through an unprotected meningeal membrane. Thank heaven all situations are not the same! The patients would have died had the surgeon not operated. I congratulate the society on this presentation, and I am sure that the members will go away with greater enthusiasm to study the subject and not allow meningitis to develop.

Dr. J. R. Lindsay: I want to thank Dr. Hagens, Dr. Pearlman, Dr. Galloway and Dr. Beck for their discussions.

In answer to Dr. Grove's question, I have shown only one view of the petrous pyramids, the so-called axial view. My associates and I also use two other views, the occiput-down view and the Stenver view. The last-named view has been favored in Europe, but in our experience the axial view has been more useful. The three views are taken in each case. If there is erosion of the superior angle the Stenver or the occiput-down view might show it best. On the other hand, suppuration in the lower part of the apex or confined within the apex shows best in the axial view.

I have purposely avoided discussing the use of sulfanilamide. We have had some experience with it during the past year but not enough for us to draw any conclusions. It may be said that our experience with this drug to date has not indicated that any of the well established surgical principles may be discarded.

This presentation has included only cases in which suppuration of the petrous pyramid was proved; in 8 of them autopsy was performed. During the eight year period in which they were collected, other cases of petrositis have also been observed and have terminated fatally. I have included all cases in which the necessary postmortem examination was possible.

Studies of Immunization of the Upper Part of the Respiratory Tract. Dr. T. E. Walsh (by invitation).

The normal defenses of the upper part of the respiratory tract have been emphasized recently by Proetz and others, who have stressed the importance of the mucous blanket and the ciliary activity as a first line of defense against infection. Various factors, both physical and dietary, combine to break down this first line of defense. Dr. Cannon and I were interested in ascertaining whether an adequate second line of defense could be set up against invasion by micro-organisms.

The results of subcutaneous vaccination against colds have been on the whole disappointing. The ideal conditions for tissue immunity are said to exist when there is an accumulation of phagocytic cells and antibodies in the tissues. These conditions can be obtained by the injection of vaccine into the tissues, as has been shown by Cannon and others. We were interested in ascertaining whether they could be brought about by vaccination of the nasal mucosa. Experiments were performed which demonstrated that after the application of vaccine of Bacterium paratyphosum B in the nose the antibody titer of the nasal mucosa is higher than it is after subcutaneous injection of the same antigen. Histologic examination of the tissue reveals a marked accumulation of large monocytes in the subepithelial tissue. Further experiments were performed, in which two different antigens were given to the same animal, one by means of intranasal application and the other by intraperitoneal injection, and the tissues and serum of the animal were titrated simultaneously against both antigens. It was found that when the serum titer is the same to both antigens the tissue titer of the mucosa and the lungs is constantly higher to the antigen given intranasally and that when the serum titer to the "general" antigen is many times higher than that to the "local" antigen the tissue titer of the respiratory tract is the same to both antigens. The explanation is offered that there is actually local formation of antibodies in the tissues of the respiratory tract after local vaccination. The conditions in the tissues after local vaccination are therefore those which are said to be ideal for tissue immunity.

Another series of experiments, in which animals were given injections of a virulent organism, were performed. After intranasal vaccination animals were immunized to several hundred lethal doses of the organism given intranasally. The probable pathway of infection after intranasal application of a living virulent organism is through the lungs. Material put in the nose is aspirated directly into the lungs. However, in unimmunized animals under anesthesia with a tracheotomy in which the upper end of the trachea was firmly plugged, virulent organisms did pass through the nasal mucous membrane and enter the blood stream, whereas similarly treated animals that had been vaccinated intranasally for five days longer were protected. The ease with which substances put in the nose are recovered from the lungs led to experiments on the effect of various types of intranasal medication on the lungs of rabbits. The slides illustrate the findings.

The clinical application of the experimental work was in prophylaxis against the common cold. A polyvalent vaccine was used as a nasal spray with encouraging results, about 80 per cent of the patients treated being free from colds, whereas in the control group 50 per cent of the patients had colds.

DISCUSSION

DR. FRANK J. NOVAK JR.: Dr. Walsh's paper is a consolidated report on work carried on over a considerable period. It is both interesting and important. It is interesting from a purely academic standpoint as a piece of excellent investigation and important because of the clinical and practical implications which it carries. I believe that the proper approach to the general problem of infections of the upper part of the respiratory tract is from an immunologic standpoint. It has been known from the work of Cannon and others that it is possible to produce local tissue immunity in the skin. After injection of antigen into the skin it is possible to measure accurately the humoral response of the skin. Moreover, it is possible to measure the cellular response to the injection. It is known that the antibody titer of that piece of tissue is increased for the time being. It is also known that there is a mobilization of various cells that have to do with the mechanism of immunity.

The result of applying that idea to the nasal mucosa has been seen. I think Dr. Walsh has perhaps investigated the humoral response, that is, the antibody response, rather than the cellular response. I like to look on the response as being primarily cellular and secondarily humoral.

If one drops a solution containing streptococci into the nose of a rabbit or a human being, one can expect to find a response to the antigen in the formation of a specific antibody. If one drops pneumococci into the nose one can expect a specific response. Practically, when one is attempting in the office or in the clinic to immunize against the organisms which are causing head colds, it is not sufficient to take one type of streptococcus, pneumococcus, catarrhal organism or staphylococcus and expect any kind of clinical result. In other words, all the evidence points away from the use of autogenous vaccines. Polyvalent vaccines containing all kinds of organisms are necessary for immunization.

I was struck with one fact in particular: While the application of a vaccine directly to the nasal mucosa produces a measurable rise in the antibody content of that mucosa, the injection over a long period will do the same thing but to a lesser extent. It seems that if one is to be practical, it might not be a bad idea to inject the vaccine to get a response from the blood and to apply it locally in the throat and nose to get a local response. I should like to ask Dr. Walsh whether any one would have the hardihood to try an injection into the nasal mucosa with a moderate amount of vaccine. It is known that the response of the skin to injections is greater than the response to surface application. If one removes the horny layer of the skin of the arm or belly by abrasion and then applies a compress of filtrate or vaccine, one gets a definite change in tissue which is directly measurable both by antibodies and by mobilization of cells that have to do with immunity. I think, however, that one gets a greater response by injecting the vaccine into the skin than by applying it on the outside, and so I wonder whether it would not be feasible to inject it into the mucosa rather than to try to raise the level of immunity by mere topical application of antigen.

I was very much interested and want to compliment Dr. Walsh for a fine piece of work.

Dr. George Shambaugh Jr.: I have used the method of intranasal vaccination against colds for two years. While I have not as yet tabulated the results, I have been impressed by them and am using the method more and more for persons who are subject to frequent colds and particularly to recurring attacks of sinus infections. I believe that this method is a real advance in the prevention of colds.

Dr. O. E. Van Alyea: It seems unusual to hear a paper on the treatment of colds by vaccines, or any paper on nasal therapy, without mention of the virus which is supposed to be the initial invader in nasal infections. I wonder whether Dr. Walsh considered this virus in his study. His work, combined with the investigations which are being carried on by Fenton and Larsell and others, should be of great value eventually. Whether or not it can be applied clinically as yet is doubtful. As Dr. Walsh stated, it is difficult to obtain authentic reports from clinic patients. Another factor is the variability according to season. I think Dr. Shambaugh's freedom from infection this season may be due to the fact that he has not yet been exposed to a severe epidemic of colds. So far this season such an epidemic seems to have been avoided.

Dr. Alfred Lewy: I should like to ask Dr. Walsh whether he noticed in his experiments that the titer of the antibodies in the lungs was as high or frequently higher than that in the nasal mucosa and how he accounts for this situation. There is a much larger epithelial surface in proportion in the lung than in the nose. Does he think that the respiratory tract reacts as a whole, or is the phenomenon purely local? Is the vaccine available for experimental purposes, and what is the cost?

Dr. T. E. Walsh: First of all I want to thank Dr. Novak for his able discussion of my paper. It has been my impression, and I believe the impression of other investigators, that antibodies are actually elaborated by histiocytes. An area of histiocytic infiltration is, I believe, necessary before vaccination will be effective. I think this was demonstrated in the experiments conducted by Dr. Cannon and me. I agree with Dr. Novak's remarks regarding polyvalent

vaccines. We controlled the specificity of the antibodies in our experiments by duplicate titrations with the homologous organisms and a control. When Bact. paratyphosum B was used in the vaccine we controlled the titrations with Bact. typhosum B, and there was absolutely no agglutination of the control organism. I think it would be somewhat heroic to inject vaccine into the nasal mucosa; as a matter of fact, we did this in one patient, and he had a marked reaction although a very small dose was injected into the inferior turbinate. He complained of severe headache and malaise and had a temperature of 104 F. I believe the injection is probably actually intravenous, as undoubtedly the large blood spaces in the turbinates are entered. I do not know just how the vaccine in an intranasal spray gets through the epithelium and into the subepithelial tissue.

Dr. Van Alyea spoke about the virus of colds. It is true that the investigations of Dochez have shown that a virus may be the causative factor. I believe its action is either to lower the resistance of the tissues or to enhance the virulence of the invading organisms which produce the symptoms of the common cold. The experimental work in the transmission of colds by means of the virus has shown that these processes are possible in only 30 or 32 per cent of the cases. The etiologic factors in the other 70 per cent are those which I discussed in my opening paragraphs.

If the immunologic reaction is so specific I think it is important to have many strains of each organism used in the vaccines. In answer to Dr. Lewy, the titer of the antibodies in the lungs of our animals was as high or higher than the titer in the nasal mucosa. This is undoubtedly because large amounts of vaccine are aspirated directly into the lungs from the nose and antibodies are presumably elaborated by the alveolar macrophages.

Book Reviews

Pathology of the Central Nervous System. A Study Based Upon a Survey of Lesions Found in a Series of Fifteen Thousand Autopsies. By Cyril B. Courville, M.D., Professor of Neurology and Psychiatry, College of Medical Evangelists, and Director, Cajal Laboratory of Neuropathology, Los Angeles County Hospital, Los Angeles. Price, $5.75. Cloth. Pp. 344, with 200 illustrations. Mountain View, Calif.: Pacific Press Publishing Association, 1937.

This is an excellent book for the clinician on the pathology of the central nervous system. The material used for its basis is 15,000 autopsies in which lesions were observed and studied, in addition to a large experience of the author with some 12,000 neurologic cases. Many of the statements made are substantiated by ample reference to works covering the subject involved. The book is not intended as a text on histologic methods for the laboratory worker, but the clinical application of the pathologic process is always stressed. The plan is good, aside from the excellence with which the book is written. The statements are clear and follow a logical sequence of events, which makes the material interesting and useful.

While histology is not a major feature of the book, it is amply covered in an instructive fashion. The author considers that all pathologic processes in the various parts of the brain are the result of the pathologic processes involving the elementary tissues composing the brain. With this as a start, structural alterations in all the tissues are considered in the introductory chapter. When specific regions are considered, it is in the light of these tissue changes, with added consideration of the specific region involved. Histology is thus briefly but thoroughly covered.

The book is divided into seven parts, in each of which a special subject is discussed as follows: (1) various anomalies and malformations, (2) diseases of the intracranial blood vessels, (3) infections of the nervous system, (4) trauma of the central nervous system, (5) intoxications, (6) diseases of unknown etiology and (7) tumors of the brain and spinal cord.

The part dealing with infections of the central nervous system should be of considerable interest to the otologist, as the various infections are considered in a logical sequence, from infections of the ear and the nasal sinuses to infections of the brain. The routes of infection are thoroughly considered, and other possible clinical manifestations are discussed. To the neurologist this should be even of greater importance, as it gives him an insight into the otologic relationship of these infections, which sometimes is overshadowed to him by the greater subject of neurology.

There is an excellent chapter on trauma of the central nervous system. The author considers the influence on the central nervous system of special incidents such as birth injuries and various injuries to the skull in traffic, as well as of injuries caused by industrial accidents. The other chapters are equally interesting. A chapter appears in the appendix on clinicopathologic aphorisms, which briefly states conclusions of clinical value drawn from the various common pathologic processes considered. There are many excellent illustrations throughout the book.

This book is an interesting one and a valuable addition to any practitioner's library. It is of value to any one who meets with organic pathologic conditions of the central nervous system. It is not merely a dry reference book; an interesting story is related about all conditions considered.

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